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CIRRHOSIS OF THE LIVER*

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CIRRHOSIS of the liver is an important, well-recognized and fairly common chronic disease, therefore I offer no apology for presenting it for your consideration. I shall give you only the cream of thirty-five years' study of this pathological condition and trust that you will find something of interest and value in it.

The pathologist uses the term cirrhosis in a broad sense. He applies it to all sclerosed conditions of the liver, whether progressive or not, in which destruction of liver cells is associated with real or apparent increase of the connective tissue stroma.

On this occasion it seems best to cover first a limited number of points which apply to cirrhosis in general and then to take up somewhat in detail the common types of cirrhosis which have interested me for many years, particularly in regard to the nature of the lesions and the causes of them.

A few words first in regard to classification. The best and simplest would be one based on etiology, as for example syphilitic cirrhosis, but this is possible at the present time to only a limited degree. For the most part the best descriptive terms available have to be used.

There are at least a dozen different types of cirrhosis but some are rare and others, like that due to the schistosoma japonicum, do not occur in this part of the world. For practical purposes we can limit ourselves to seven different types and I shall discuss and illustrate each of them so that you will be able to carry away a fairly clear idea of how they look under the microscope and to some degree in gross.

An analysis of all the cases of cirrhosis of the liver which have come to postmortem examination at the Boston City Hospital during the past thirty-five years (1897 to 1931 inclusive) shows the frequency of occurrence of the different types in this part of the world. No case of streptococcus cirrhosis, of which I shall speak later, was recognized although the lesion was studied in several livers from other sources.

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One point must be borne in mind in connection with these statistics. Of the 9346 autopsies 2066 were made on children, mostly from the infectious wards (scarlet fever, diphtheria, etc.).

TABLE 1

CASES OF CIRRHOSIS AT THE BOSTON CITY HOSPITAL

Total number of autopsies during the past 35 years		9346	
Total number of cases of well-marked cirrhosis of the liver		550	5.88%
They are classified as follows:			
		No.	%
1.	Alcoholic cirrhosis	270	48.90
2.	Pigment cirrhosis	49	8.90
3.	Healed acute yellow atrophy	46	8.36
4.	Syphilitic cirrhosis	28	5.09
5.	Colon bacillus cirrhosis	25	4.54
6.	Obstructive cirrhosis	24	4.36
7.	Obstructive and colon bacillus cirrhosis	3	0.54
8.	Cancer cirrhosis	1	0.18
9.	Not classified	104	18.90

The cases of cirrhosis listed as not classified represent late or healed stages of various types (alcoholic, pigment, healed acute yellow atrophy) which can no longer be recognized with certainty.

TABLE 2

AGE

Decade	Alcoholic	Pigment	Healed A. Y. A.	Syphilitic	Colon bacillus	Obstructive	Colon bacillus and Obstr.	Cancer	Not Classified
1			2	2					
2			5	1					1
3	9		4	2	3				1
4	54	5	4	4	4	1			9
5	78	10	8	11	5	1		1	19
6	76	17	12	5	4	7			29
7	40	13	7	3	9	8			33
8	11	4	4			6	2		7
9	2					1	1		5
Total	270	49	46	28	25	24	3	1	104

Table 2 shows the age at which the different varieties of cirrhosis are most likely to occur.

Certain types (healed acute yellow atrophy, syphilitic) may occur at any age while others (alcoholic, pigment) are practically limited to adult life being dependent largely on the imbibing of alcoholic liquors and on occupations.

The most important point evident in the table is the late age at which alcoholic and pigment cirrhoses cause death, indicating that the lesions require many years for development.

TABLE 3

SEX

Type of cirrhosis	Males	Females	Total
Alcoholic	171	99	270
Pigment	44	5	49
Healed acute yellow atrophy	29	17	46
Syphilitic	15	13	28
Colon bacillus	18	7	25
Obstructive	14	10	24
Colon bacillus and obstructive	3	0	3
Cancer	1	0	1
Not classified	70	34	104

Table 3 shows how frequently the two sexes were affected in this series. It must be borne in mind, however, that males are probably more likely than females to go to a hospital for treatment. Moreover, drinking and social habits and the trades and occupations of men play an important part in increasing their proportion. In this series of alcoholic livers it was noticeable however that the majority of the most active lesions occurred in women.

These two conditions are considered together because they are so often combined. The table shows the frequency of occurrence in the different types.

One point of interest shown in this table is the presence of ascites in colon bacillus cirrhosis (so-called Hanot's cirrhosis). It is commonly stated to be absent. Most cases to which clinically the term Hanot's cirrhosis is applied prove on postmortem examination to be the early fatty stage of alcoholic cirrhosis.

TABLE 4

ASCITES AND JAUNDICE

Type of cirrhosis	No. of cases	Ascites alone	Jaundice alone	Ascites and Jaundice combined	Total
Alcoholic	270	48	40	53	141
Pigment	49	9	12	11	32
Healed acute yellow atrophy	46	10	15	8	33
Syphilitic	28	6	5	2	13
Colon bacillus	25	4	9	8	21
Obstructive	24	1	10	6	17
Obstructive and colon bacillus	3			1	1
Cancer	1			1	1
Not classified	104	17	5	9	31

TABLE 5

ESOPHAGEAL VARIX AND HEMORRHAGE

Type of cirrhosis	No. of cases	Varix	Hemorrhage
Alcoholic	270	24	20
Pigment	49	2	2
Healed acute yellow atrophy	46	3	3
Syphilitic	28	2	1
Colon bacillus	25	1	1
Obstructive	24	1	1
Obstructive and colon bacillus	3	0	0
Cancer	1	0	0
Not classified	104	12	8

This table shows the different types of cirrhosis in which varix and hemorrhage occurred. It is probable that the actual number of cases of varix would have been much larger if a careful examination had been made for them in every instance.

The largest alcoholic liver uncomplicated by fat weighed 2990 grams. The enlargement of others was due to the presence of fat which caused the largest liver to weigh 5335 grams.

The largest of the pigmented livers weighed 4350 grams and was known to be enlarged for twenty years before death. No fat was present.

TABLE 6

LIVER—WEIGHT IN GRAMS

Type of cirrhosis	1 to 500	501 to 1000	1001 to 1500	1501 to 2000	2001 to 2500	2501 to 3000	3001 to 3500	3501 to 4000	4001 to 4500	4501 to 5000	5001 to 5500
Alcoholic		3	67	87	55	34	13	6	1	1	2
Pigment		1	18	13	8	4	2	1	1		
Healed A. Y. A.		21	12	8	3			1			
Syphilitic	2	1	8	12	6	1					
B. coli		3	13	5	2	1	1				
Obstructive			8	11	4						
Obstr. and B. coli			1		1						
Cancer						1					
Not classified		7	39	35	17	2			2	2	

In the unclassified series the large weights were due to the presence of fat.

The smallest livers in the alcoholic series weighed as low as 985 grams, in the pigment series 1015 grams and in the healed acute yellow atrophy series 510 grams; other low weights, all in adults, in this last group are 595, 705, 712, 817, 840, 860 grams.

The small syphilitic livers were in infants.

TABLE 7

SPLEEN—WEIGHT IN GRAMS

Type of cirrhosis	1 to 150	151 to 300	301 to 400	401 to 500	501 to 600	601 to 700	701 to 800	801 to 900	901 to 1000	1001 to 1100	1101 to 1200	3990
Alcoholic	73	112	43	18	12	5	4	2	1			
Pigment	8	19	10	6	5			1				
Healed A. Y. A.	17	11	6	4	3	2	1	1			1	
Syphilitic	9	10		2	2	2				1		
B. coli	9	7	2	3	1	2			1	1		
Obstructive	9	9	3	1		1						
Obstr. and B. coli	1	1										
Cancer	1											
Not classified	34	39	15	4	6		1	1	1	2		1

Enlargement of the spleen in cases of cirrhosis is of frequent occurrence and is due to obstruction to the flow of blood through the liver. The effect of the back-pressure in the portal system is to cause a dilatation of the vessels of the spleen and a stretching of the stroma. As a result of the mechanical injury the fibroblasts proliferate and produce an increased amount of collagenous and elastic fibrils thereby effecting a distinct sclerosis of the organ.

The size of the largest spleen listed (3990 grams) was due to congestion and multiple infarcts.

TABLE 8

PRIMARY LIVER CELL CANCER

Alcoholic cirrhosis	3
Pigment cirrhosis	4
Healed acute yellow atrophy	3
Colon bacillus cirrhosis	1
Unclassified	1
Total	12

An important and not very infrequent complication of cirrhosis of the liver is primary liver cell carcinoma which rarely occurs under any other condition. The constant regeneration of liver cells results once in so often in the freeing of a cell from normal control and it then proceeds to grow as a tumor. The growth may produce great increase in the size of the liver and occasionally gives rise to metastases to regional lymph nodes and to the lungs. When present it is usually the immediate cause of death. Table 8 shows the types of cirrhosis at the Boston City Hospital in which liver cell cancer occurred.

EFFECTS OF CIRRHOSIS

In order to understand the injurious mechanical effects of sclerosis of the liver two important points must be borne in mind.

a) The fibroblasts of the stroma of the liver proliferate under four conditions; (1) to replace any which have been destroyed as in abscesses, infarcts, etc.; in colon bacillus cirrhosis; and in syphilis; (2) following mechanical injury (stretching) as in chronic passive congestion and bile stasis; (3) to organize fibrin (thrombi in sinusoids); and (4) to form stroma for islands of regenerated liver cells and for tumor nodules.

b) The collagenous and elastic fibrils produced by fibroblasts tend to contract unless they are kept stretched by the presence of liver cells or by dilated blood vessels, and these two elements are not effective if the fibrils are present in excess of normal conditions.

Contraction of connective tissue in cirrhosis of the liver has two injurious effects; it impedes the flow of blood through the organ and may block the outflow of bile more or less completely. Obstruction of the circulation, especially in the portal system, causes chronic passive congestion of the gastro-intestinal tract with increase of mucous secretion from the glands (chronic catarrh); mechanical enlargement of the spleen owing to distention of the venous sinuses; escape of fluid into the peritoneal cavity (ascites); and dilatation of various veins in an attempt at establishing collateral circulation. Of the distended veins those in the abdominal wall forming the caput medusae are the most conspicuous but those in the esophagus and gastric cardia the most to be feared owing to the danger of erosion followed by hemorrhage and death.

Obstruction of the bile ducts in cirrhosis is not general, as when a stone or cancer blocks the common duct, but focal; it may, however, be widespread and involve large parts of the liver so that much bile passes into the circulation and gives rise to intense jaundice. The occurrence of ascites and jaundice in connection with the various types of cirrhosis has certain peculiarities not all of which can be explained.

In alcoholic cirrhosis either ascites or jaundice may appear first or both may come on at the same time. The same is true of pigment cirrhosis.

In obstructive cirrhosis jaundice appears at once owing to the nature of the lesion. It usually occurs early also in colon bacillus cirrhosis following cholangitis, either because the exudate blocks the escape of bile or, perhaps more likely, because this type of lesion is a common complication of obstructive cirrhosis.

In acute yellow atrophy, jaundice is an early and characteristic sign owing to destruction of liver cells and interruption of the flow of bile in the bile capillaries. After regeneration of the liver cells has taken place, contraction of connective tissue in those foci where no regeneration has taken place may eventually (sometimes only after many years) lead to obstruction of bile ducts and blood vessels. The same holds true in syphilitic cirrhosis following organization of gummas and contraction of the scar tissue.

1. OBSTRUCTIVE CIRRHOSIS

In the table giving an analysis of the cases of cirrhosis occurring at the Boston City Hospital during the past thirty-five years, the different types were arranged according to their frequency. In discussing them I shall take them up in a different order beginning with obstructive cirrhosis which is of simple mechanical origin. It is caused by occlusion of either the hepatic or the common bile duct, usually by calculus or cancer. The best example of this lesion is seen perhaps in cases of congenital cirrhosis in infants due to lack of development of the common duct because there is no chance of complication by secondary infection.

When bile stasis takes place the bile ducts become dilated, bile thrombi form in the bile capillaries, and bile often accumulates in the form of granules and spherical masses in the cytoplasm of the liver cells. Dilatation of the bile ducts leads to stretching of the surrounding fibroblasts and to a proliferation of them, resulting in a periportal increase of stroma which may be considerable in amount. This type of cirrhosis is often called biliary owing to the location of the lesion.

The bile droplets in the liver cells may become decolorized and persist as globules which stain intensely with eosin, suggesting the hyalin of alcoholic cirrhosis. The bile thrombi often break out of the bile capillaries and are taken up and dissolved by monocytes. The bile in the bile ducts may form concretions, become surrounded by monocytes and foreign body giant cells and be dissolved or eventually become organized by fibroblasts just as fibrin does in other locations.

The lesion of obstructive cirrhosis is often complicated by small infarcts in the peripheries of the lobules owing to obstruction of blood vessels by the dilated ducts. Organization of these infarcts adds to the sclerosis present.

A patient with complete bile stasis can live for weeks or months (an adult 9 months, an infant with congenital bile stasis 4½ months). If the obstruction is removed the liver tends to return to the normal condition but any increase of stroma persists. The result is a slight to a considerable sclerosis around all the bile ducts. The condition is very evenly distributed throughout the whole liver. The result is a smooth to finely granular type of sclerosis which is commonly called biliary cirrhosis. A better and more distinctive term is "obstructive cirrhosis" as it indicates the method of origin of the lesion and does not confuse it with colon bacillus cirrhosis which is also commonly included under the term biliary cirrhosis.

2. COLON BACILLUS CIRRHOSIS

Another type of cirrhosis must be considered in connection with the obstructive and that is the one due to infection of the bile ducts by the colon bacillus. It frequently complicates the first owing to the fact that obstruction favors infection. The lesion starts as an ascending cholangitis and like the obstructive involves the bile ducts and surrounding tissues. It usually extends more or less diffusely all through the liver. The bile ducts become distended with an exudation of serum and polymorphonuclear and endothelial leucocytes in various proportions. The lining epithelium is often destroyed in places. From the bile ducts the bacilli invade the periportal connective tissue destroying fibroblasts and causing an inflammatory exudation so that the portal tissues show up as broad bands. In many places the lesion encroaches on the adjoining liver cells which degenerate and undergo necrosis. Invasion and dissolution by leucocytes follow. In this way the lesion spreads more or less irregularly towards the hepatic vein.

In many places the inflammatory exudation in the smaller bile ducts leads to occlusion of them and to obstruction to the outflow of bile, which appears in the ducts above the point of obstruction in the form of yellowish-green inspissated masses. The obstruction is focal only, however, not general, and this accounts for the moderate degree of jaundice produced.

This type of infection is likely to become chronic so that much destruction of liver cells and connective tissue may occur followed by regeneration of fibroblasts. The situation of the lesion chiefly around the portal vessels seems to be fairly characteristic.

In the acute stage the liver is enlarged, sometimes to double the normal size, and the surface is smooth. If any type of cirrhosis should be recognized as Hanot's, it would seem to be the early stage of this. In the healed stage the organ may be below normal in weight and present a finely granular appearance. On section

the cut surface shows a monolobular arrangement owing to the lesion having been located at the periphery of the lobules.

This type of cirrhosis is fairly rare and occurs not only in childhood, but also at other times of life and even in old age. In the early stage it is attended with fever, jaundice and often with rapid enlargement of the liver. In the late or healed stage it may be difficult or impossible to distinguish between an obstructive or colon bacillus cirrhosis or a combination of them. For this reason the term biliary cirrhosis is useful to apply to either of them or to the combination.

3. HEALED ACUTE YELLOW ATROPHY

There is one type of cirrhosis which clinicians in general do not seem to recognize and that is the one which follows acute yellow atrophy. It is not a chronic progressive process although, as Dr. J. H. Pratt has shown, it may be recurrent. The lesion consists of sudden extensive necrosis of liver cells, followed by their solution and removal by the action of the enzymes of invading leucocytes. The milder degree of the process is known as central necrosis and is relatively common. The cells may regenerate or be followed by central sclerosis. In chronic passive congestion, regeneration, which ordinarily does not take place, is characteristically irregular in distribution suggesting a fern leaf appearance. If, as in acute yellow atrophy, all the cells in a lobule are killed no regeneration of liver cells in that lobule can take place because, after fetal life, they arise only from liver cells. The bile ducts grow out about half way towards the center of the lobule and then stop. In time the stroma contracts and sclerosis results.

If liver cells are left alive either at the periphery or scattered here and there in the lobule, active regeneration takes place and the lobule is rebuilt or large nodules of liver cells are formed without lobular arrangement.

The gross appearances of the acute yellow atrophy liver found postmortem vary greatly, according to the length of time which intervenes between the liver-cell necrosis and the death of the patient. At the end of about one week the liver cells have to a large extent been dissolved and removed. Their place is occupied by a variable number of endothelial leucocytes. The liver has shrunken to one-half or even almost to one-third of its normal size (595 and 600 grams respectively in two instances). Here and there, especially around the portal vessels, single living liver cells and small groups of them may be found. In gross the liver is uniformly markedly diminished in size. Its edges are thin and its surface is delicately wrinkled. It is noticeably flaccid and can be easily rolled upon itself. Its color is yellowish red.

In case the patient lives two to four weeks or more, regeneration of the liver cells plays an important part and alters the gross appearance of the organ. In those parts of the liver where the hepatic cells remain uninjured around the portal vessels, the lobules may be completely regenerated and distended up to their normal size again (or even beyond that as the result of compensatory hyperplasia). In other lobules where only one or more cells remain uninjured, only small clumps of cells result from regeneration. In those areas where necrosis of all the liver cells took place, the shunken lobules persist with a network of capillary-like vessels at the center around the hepatic veins, and small bile ducts stretching out towards them from the portal vessels at the periphery.

In consequence of these regenerative changes a liver at this stage of the process looks as though only a part of it had originally been injured and it weighs more than at the earlier stage (960 grams in one instance). The regenerated areas appear of an intense yellow hue due to bile staining of the regenerated liver cells. The atrophied portions are of a red color, owing to little else remaining than sinusoids filled with blood.

If the patient recovers after extensive necrosis and acute atrophy of the liver the shrunken portions in time become more or less completely sclerosed owing to disappearance of the sinusoids and contraction of the connective tissue. Such a liver when found postmortem is classed under the general heading of cirrhosis of which it forms an interesting and fairly common type. It represents the healed stage following sudden widespread necrosis of liver cells.

In typical early cases the recognition of this type of cirrhosis is easy because in the atrophied portions the characteristic markings of the lobules are perfectly preserved. At a later stage they may be pulled and stretched all out of shape by the regenerated parts in which usually no definite lobular arrangement is evident.

The gross appearance of the liver of healed acute yellow atrophy is fairly characteristic. It is considerably diminished in size and usually coarsely and irregularly granular and nodular. The nodules may measure up to one or two or more centimeters in diameter. The atrophied portions are depressed, finely granular and often many centimeters in extent. The left lobe is usually more affected than the right. Occasionally there are present multiple spherical islands of regenerated liver cells looking like adenomas and showing microscopically no bile ducts and no sign of lobular arrangement.

This type of cirrhosis occurs at all ages and is relatively common in young people. It is frequently mistaken for alcoholic cirrhosis, which it sometimes closely resembles, and more than once has been diagnosed by the surgeon, on account of its nodular appearance, as metastatic tumor of the liver.

Acute necrosis of the liver is caused by a

variety of bacterial toxins, especially those derived from the streptococcus and pneumococcus. It is also often due to poisons such as arsenic, phosphorus, chloroform and carbon tetrachloride.

4. STREPTOCOCCUS CIRRHOSIS

It seems probable that acute yellow atrophy is sometimes produced by the direct action of a streptococcus in the lesion instead of by toxins and poisons brought to the liver through the circulation in the manner just described.

The best examples of the lesion have been found in infants a few days after birth as the result of infection of the umbilical cord followed by extension of the process to the liver; but a similar type of lesion may occur in adults owing to streptococcus septicemia.

In these rare cases the streptococcus causes necrosis of only the parenchymatous cells. The process may involve small or large areas and be more or less uniformly distributed, suggesting a zonal necrosis, or occur in sharply defined foci. The organism dies out quickly, the necrotic liver cells are dissolved and removed through the action of leucocytes and the final result is variously sized areas of acute red atrophy which shrink and in time appear as foci of sclerosis. If the original infection was more or less extensive the liver eventually presents the appearance of an atrophic cirrhosis characterized on the surface by elevated nodules of all sizes composed of uninjured or of regenerated liver cells and by small and large depressed areas in which no liver cells are present. The lesion forms a rare type of cirrhosis following repair after acute red atrophy of infectious, not of toxic, origin.

5. SYPHILITIC CIRRHOSIS

Syphilitic infection of the liver is usually divided into two types, the congenital and the acquired. In the congenital type a diffuse lesion predominates, in the acquired a focal lesion, but the two types are often combined in the congenital form and may coexist to some extent in the acquired.

In congenital syphilis the treponema pallidum causes injury to the fibroblasts of the stroma and a slight inflammatory exudation of endothelial leucocytes. As a result of the active and continued regenerative proliferation of the fibroblasts the connective tissue increases greatly in amount. The relation of organism to lesion is shown by the fact that where the treponemas are most numerous the connective tissue is most abundant. Later, as the collagen fibrils contract the liver cells are compressed and more or less atrophy of them accordingly results. This diffuse type of lesion, resulting in primary increase of connective tissue and secondary atrophy of liver cells, is perfectly well recognized as characteristic of congenital syphilis. It is not infrequently complicated with focal lesions (miliary and larger infarctions, commonly called gummas) due to occlusion of arteries by syphilitic endarteritis obliterans.

In acquired syphilis the lesion is rarely or never distributed uniformly throughout the organ but affects larger or smaller foci only and leads usually, perhaps always, to the formation of gummas so characteristic of this type of infection. They appear as single and multiple nodules of various sizes in the substance of the organ and projecting from its surface. Following organization by capillaries and fibroblasts and by contraction of the resulting dense fibrous tissue they are replaced by radiating scars and by deep depressions which frequently divide the liver into multiple lobes of all sizes, whence the origin of the term hepar lobatum characterizing the condition but not necessarily diagnostic of it.

Gummas occur most frequently in the left lobe and in the region of the suspensory ligament.

6. PIGMENT CIRRHOSIS

Pigment cirrhosis forms the first and most important lesion in the disease known as hemochromatosis. It is characterized by the presence of two pigments in the liver cells, hemofuscin derived from hemoglobin and hemosiderin which is slowly developed from the hemofuscin and is usually much the more abundant. In time the liver cells undergo necrosis. The pigments are taken up by endothelial leucocytes which accumulate in the stroma, chiefly in the periportal connective tissue. Regeneration of liver cells takes place in the form of small nodules up to 1 or 2 mm. in diameter. The new liver cells at first are non-pigmented. Gradually hemofuscin is deposited in them and slowly changes to hemosiderin. These cells may in turn undergo necrosis. They are killed, not by the pigment, but by the injurious agent which leads indirectly to the deposit of the hemofuscin.

Hemofuscin is deposited also in the endothelial cells lining the sinusoids, and much more slowly in the bile duct epithelium, in fibroblasts of the stroma, and in the smooth muscle cells of the veins and arteries. These various types of cells, however, do not undergo necrosis.

Where the liver cells are killed off the stroma contracts and condenses, thus forming small foci of sclerosis. New stroma is formed only in the islands of regenerated liver cells and it in turn may eventually become a patch of sclerosis. In this way the amount of connective tissue in the cirrhotic liver becomes increased.

The gross appearance of the liver is fairly characteristic owing to the light to dark brown,

occasionally chocolate, color caused by the presence of the pigments. Congestion may mask it to some extent but the ferrocyanide of potassium reaction performed on a thin slice of fresh material will determine the presence of iron within a few minutes. A brownish color of the pancreas and of the adjoining lymph nodes helps to confirm the diagnosis.

In the early stages of the process the liver may be increased in weight (2930, 3280, 4350 grams); later it diminishes in size and may go as low as 1260 grams.

The surface gradually becomes granular as in alcoholic cirrhosis, never coarsely nodular, and its consistence is steadily increased. Occasionally the islands of regenerated liver cells are plainly evident, standing out as small white nodules up to 1 or even 2 mm. in diameter in sharp contrast with the brown background.

Pigment cirrhosis is not so rare as commonly supposed, forming about 9 per cent of all the cirrhoses in our series and is often combined with the alcoholic.

It is much more common in men than in women (44 men, 5 women in our series), is extremely rare as a cause of death before the age of thirty and increases in frequency from then up to sixty years of age. It forms the chief lesion in the chronic disease named hemochromatosis by von Recklinghausen and characterized by pigmentation of the liver, pancreas, heart, skin and various other organs.

Parker, Nye and I were able to show that it is possible to produce pigment cirrhosis in rabbits, sheep and monkeys by means of chronic poisoning with copper in 6 to 12 months provided the dose is large enough.

Recently Parker and I were able to demonstrate copper in the islands of regeneration in human livers by means of a very delicate microchemical test. Pure hematoxylin in a neutral solution stains copper a clear blue while the iron present in hemosiderin is colored black after alcoholic fixation or yellow to brown after formalin fixation.

Our experimental work shows that copper has a twofold action. It injures liver cells causing cirrhosis, and destroys red blood corpuscles setting free the hemoglobin which is deposited in the liver and later in the other organs as copper hemofuscin. The copper is quickly eliminated through the bile leaving hemofuscin which gradually changes to hemosiderin. The striking and characteristic features of the disease are therefore a secondary affair due to hemoglobin, not to the primary effect of the copper. In man, so far as can be ascertained from clinical observation, well-marked pigment cirrhosis requires in the neighborhood of fifteen years for development.

The chief sources of chronic poisoning are to be found in distilled liquors as a result of the use of copper coils in the distilling apparatus; in wines made from grapes obtained from vineyards where copper salts have been sprayed on the vines as insecticides; and in occupations involving the cutting, grinding and polishing of copper and brass pipes, rods, utensils and instruments.

7. ALCOHOLIC CIRRHOSIS

We come now to the commonest type of cirrhosis, that known as alcoholic cirrhosis on account of its intimate association with excessive indulgence in alcoholic beverages. All experimental evidence is against alcohol being the cause of this lesion which is probably due to some substance occasionally contaminating liquors. Rarely it may be taken into the body in other ways.

It is necessary in the first place to describe the peculiar form of lesion which occurs in this type of cirrhosis because it is characteristic and diagnostic of it and is not generally recognized. The cytoplasm of the liver cells very slowly undergoes a degenerative change in consequence of which minute hyaline droplets appear in it and gradually enlarge and fuse to form an irregular fine to coarse hyaline meshwork which tends to surround the nuclei. This meshwork or reticulum, except when very old, stains intensely with eosin in the eosin methylene blue method after fixation in Zenker's fluid. The degenerative change may attack single cells or small or large groups of them. It may affect cells throughout the lobule but if slight in amount is confined chiefly to the center of it in the early stage of the process.

As the liver cells in time are killed off regeneration takes place, often in the form of small islands 1 to 2 mm. in diameter. In these islands new stroma is formed. In this way the connective tissue of the liver is gradually increased in amount. In those foci where regeneration of liver cells does not take place the stroma condenses and sclerosis results. This type of cirrhosis is very commonly complicated by fatty infiltration, especially in its earlier stage.

As may be imagined from the changes taking place in the liver in alcoholic cirrhosis, the gross appearances vary greatly. The size may run from three kilograms, or if fat is present from five kilograms or more, down to one kilogram or even slightly less. The surface is smooth in the early stage but gradually becomes more and more granular to finely nodular as the connective tissue increases in amount, contracts and leaves the old or newly formed liver cell masses projecting.

The color is not characteristic. The normal liver is light brownish. Fat in the cells turns it yellow. The connective tissue of the stroma is

grayish; congestion adds red and bile stasis yellow and brown to green. The result is often a marked combination of colors.

The shape of the liver is usually not altered. Hypertrophy and atrophy occur symmetrically. The edges are rounded when the liver is large and thin and granular or finely nodular when it diminishes in size.

The consistence steadily becomes greater as the relative amount of connective tissue increases, so that the liver cuts with resistance, is dense and incompressible to the touch and is torn with difficulty.

The lesion is of fairly frequent occurrence, affects women as well as men, although more rarely, and occurs most often in the fourth, fifth and sixth decades.

Very rarely the lesion may occur in childhood and sometimes, so far as can be judged from clinical evidence, may affect 2 and even 3 members of the same family. I have personally studied the lesion in the livers of four children between the ages of 10 and 17 years: two of them were brother and sister.

I wish now to say a few words about the experimental production of this type of cirrhosis. Over thirty-five years ago, when I first took charge of the pathological laboratory at the Boston City Hospital, I saw two marked examples of alcoholic cirrhosis, one very extensive, the other much less so. The microscopic lesion in both was so striking and distinctive that I felt there must be a definite cause for it. I have always regarded the lesion as a finger print which, as in a criminal case, would lead to the discovery of the agent which caused it. It was purely morphological but it was the only evidence available.

Like many others I have tried to produce the lesion by various kinds of alcohols, but without success. For the past eighteen years the search for the cause has been an obsession. I collaborated first with Dr. McJunkin, then with Drs. Parker and Nye, all assistants in the laboratory. Of late I have continued the search alone. Everything was tried that could be conceived of as possibly contaminating liquors such as twelve different metals and at least twenty-eight other substances including various alcohols, furfurol, creosote, turpentine, oil of juniper, etc.

My own efforts for several years have been devoted chiefly to lead and arsenic. Both were promising when used in large doses, but given in small amounts over long periods of time they led to nothing. It was impossible to produce chronic lesions in the liver by the administration of either of them.

For over two years I have been trying another agent which is used therapeutically and might explain the occurrence of the lesion in children, but which one would never imagine contaminating alcoholic beverages. Experiments performed during the past four months have shown that used in extremely minute doses, 1 milligram to 1 kilogram of body weight, it produces cirrhosis of the liver in rabbits and guinea pigs in eight to twelve weeks with more or less marked bile stasis and occasionally with slight ascites. The liver cells contain numerous fine to coarse droplets which stain deeply with eosin, by the eosin-methylene blue method after fixation in Zenker's fluid. The granules are grouped around the nuclei and in many cells tend to fuse into a coarse reticulum, but in this limited time have not formed definite hyaline material. Evidently the dose is too large and the time too short. The cells are killed off rapidly and regeneration is very active. Small foci of new liver cells are found everywhere. The cells are being killed too quickly to permit the production of the complete degeneration desired. In human beings the degenerative process is very much slower, occupying months to years instead of weeks.

The fibroblasts of the stroma are very active, especially around the portal vessels. Apparently they are stimulated to proliferate, probably as the result of injury produced by the agent employed. Bile duct proliferation is also active.

Over a year ago I found that the eosinophilic granules and reticulum in the degenerating human liver cells in alcoholic cirrhosis would give a definite microchemical reaction with certain colorimetric tests. The same tests applied to the eosinophilic granules in the cells in the cirrhotic livers produced in animals gave positive results.

The substance which is such a destructive agent for liver cells even when used in extremely minute quantities over a considerable period of time and which produces a characteristic type of degeneration is phosphorus.

Many years ago I was called over to the surgical floor by Dr. E. H. Nichols. He was operating for obstruction in a young woman on whom appendectomy had been performed a year or two previously. The intestines were all glued together by fibrous adhesions and here and there in the adhesions were small solid masses about the size of peas. He cut open one of the nodules which was soft within and asked me what it was. "Vaseline", I said. He threw up his hands in disgust because he realized at once how it had got there. I didn't. In those days oil was sometimes poured into the peritoneal cavity with the idea of preventing adhesions. Unfortunately the earlier operator had used vaseline, which acted as a foreign body and produced the very adhesions he was seeking to avoid.

Later I asked two or three surgeons who had been present at the operation if they couldn't

recognize vaseline when they saw it. "Oh yes," each one answered, "but I couldn't see how it could get there."

I am in the position of those surgeons. I can't see how phosphorus can get into alcoholic liquors unless deliberately put there or derived from phosphides in cast iron containers, but that isn't going to prevent my saying that as the result of positive Macallum and Fiske-Subbarow microchemical tests for phosphorus and the experimental production of a characteristic cirrhosis of the liver in rabbits and guinea pigs by the administration of phosphorus, it is conceivable that alcoholic cirrhosis may be due to chronic poisoning with phosphorus. However, certain desirable links in the chain of proof are still missing so that I do not feel justified in doing more than advancing this idea as a suggestion. One point in its favor is the fact that the same type of lesion occurs under rare conditions in children to whom (as well as occasionally to adults) phosphorus is sometimes administered in oil or as an elixir to stimulate the growth of bone as in rickets and certain other pathological conditions. Judging from experimental evidence, phosphorus must certainly be regarded as an exceedingly dangerous remedy to use.

In conclusion it is only fair to acknowledge that very few people will take any stock in this theory until the occasional presence of phosphorus in alcoholic beverages has been demonstrated. That is evidently a problem for the chemist.

"TOO SMALL TO PICK BERRIES"

The approach of the season for the employment of migratory workers in berry and vegetable fields gives interest to the story of the children's nursery and kindergarten maintained last summer in Brant, N. Y., as told in the *Federal Council Bulletin*. This center—the first of the kind in New York State—was opened for the children "too small to pick berries" who come yearly to the neighborhood with the migrant workers. It was made possible through the coöperation of the Council of Women for Home Missions, church women of the neighboring communities, and from all over the State, the growers, and the local school board. Baby clothes, rompers, kitchen utensils, nursery supplies including baskets to be used as babies' cribs, and kindergarten equipment were donated or lent. The house was painted, papered, made sanitary and prettily curtained, ready for the 63 children who stormed it on July 1. They ranged in age from 6 months to 8 years.

The county nurse made regular visits to inspect the children and give emergency treatments and to advise the council workers. Girl Scouts assisted, thereby earning their child-nursing badges. Neighbors gave friendly aid. A midday meal and an afternoon lunch helped many a little "bag of bones and nerves" to gain height and strength.—*U. S. Children's Bureau.*

O SAY, CAN YOU SEE?

Under the title "O Say, Can You See?" the National Society for the Prevention of Blindness has made of its report for 1931 an illustrated appeal for increase of the sight-saving work which it is sponsoring. It states that in 1931 only 7.5 per cent of new admissions to schools for the blind were victims of babies' sore eyes, a decrease from 28 per cent in 1907. This cause of blindness, it points out, can be entirely eradicated by preventive treatment of the eyes of every new-born baby. The society reminds us of the dangers to children's eyes that lurk in firecrackers, air guns, sharp-pointed toys, and in scissors, forks, and knives used as playthings.

During the year the national society coöperated with Columbia University in a study of the actual effects of reading on the eyes and in the training of teachers for sight-saving classes; with the National Education Association and the American Medical Association in work for conserving the sight of school children; and with the Illuminating Engineer Society in determining lighting standards for schools. During the year 22 additional sight-saving classes were organized, bringing the total number in the United States to 398. These were in 114 cities in 23 different States.—*U. S. Children's Bureau.*

CAMPAIGN AGAINST SMOKE

Steps have been taken in New York City recently to organize a National Smoke Abatement Campaign because of the growing interest in this particular problem shared by all large cities. Up to the present time more than 100 ordinances for the prevention of smoke have been enacted. The argument for more effective action against the smoke nuisance is usually based on public health considerations. Investigations which have been continued by the Mellon Institute and other agencies have revealed that there are definite risks in the inhalation of smoke-polluted air. Added to the health feature, smoke is a matter of important economic interest in that it is destructive of many forms of property and increases to a marked degree the expense of maintaining cleanliness.

A SUGGESTION FOR AN INHIBITOR OF CANCER

Under date of May 20 the Rockefeller Institute suggested the possible discovery in embryonic skin of a substance that can inhibit cancerous growth. The explanation is that this new agent may be a part of the mechanism long sought by science which stops growth of any part of the body at just the right time. For example, any member of the body ceases growth under normal conditions at a time when it has attained its relative power in the maintenance of the functions of the body as a whole. Along this line a growth inhibitor has been found to exist in chicken cancer which inhibits the growth of mouse cancer. In addition to the existence of this substance in embryonic skin it is believed to exist also in the placenta.

THE OPERATIVE MORTALITY IN GOITRE*

BY HOWARD M. CLUTE, M.D.†

IN this country the surgical removal of abnormal thyroid tissue is now well established as the treatment of choice in certain diseases of the thyroid gland. Most physicians are coming to feel that undue delay in the application of adequate surgery in hyperthyroidism is as much to be deprecated as unjustifiable delay in the use of surgery in acute appendicitis. With increasing frequency in recent years, it is becoming recognized that the single and multiple thyroid adenomata should be removed surgically for the prevention of malignancy in goitre and that adenomatous goitres should be removed for the cure or the prevention of hyperthyroidism and the relief of pressure symptoms. It is essential, in order that this attitude as to the value of surgery in thyroid disease shall be sound, that surgeons who deal with thyroid patients in large numbers shall demonstrate the safety of the surgical treatment of thyroid disease.

In reviewing the surgical experience of this Clinic with thyroid disease in the year 1931, we are impressed with the safety now attained by surgery in the treatment of goitre. This is shown by the fact that 1197 operations were performed on 993 patients with goitre in the past year in the Clinic. Eight of these patients died, giving a mortality of .8% or an operative mortality of .66%.

It is quite properly assumed that, in the absence of hyperthyroidism, thyroid surgery today should carry with it little or no mortality. We feel very strongly that in non-toxic adenomata of the thyroid, either single or multiple, in thyroiditis and in simple goitres there should rarely be any operative mortality. That this result is possible is evident since there was but one death in three hundred and ninety non-toxic, single or multiple adenomatous goitres operated upon in 1931—a patient mortality of .25%. This patient who died had a coronary embolus six days after operation.

Fifteen patients were operated upon during the year for thyroiditis of various types and degrees. There were no deaths. It is interesting to note that ten of these patients had simple thyroiditis, one patient had a definite abscess formation and four patients had Riedel's struma. Thyroiditis resulting in abscess formation has been very rare in our experience, being limited to about six cases in all the thyroid work of the Clinic. In this patient excellent recovery followed partial resection of the lobe containing the abscess and no clinical evidence of abnormal thyroid function followed the procedure.

Cancer of the thyroid is appearing more frequently in our recent experience than heretofore, due, we believe, to the increased diligence with which Dr. Shields Warren and his associates in the pathological laboratory search adenomatous goitres of all types for evidence of blood vessel invasion. This one histological factor, as pointed out by Dr. Allen Graham, is itself sufficient to establish a diagnosis of potential malignancy in an otherwise apparently benign tumor of the thyroid. The mortality associated with the surgical treatment of thyroid malignancy will vary exactly as one attempts or eliminates operation in the serious and advanced cases. We have become more conservative in dealing with thyroid malignancy as our knowledge of the condition increases. We have found that a very definite mortality accompanies simple biopsy on thyroid cancers which have obviously become totally inoperable. Our tendency, therefore, is to omit biopsy on these tumors that can be diagnosed readily on clinical examination in which thyroidectomy is impossible and to give them x-ray treatment at once. In 1931, however, we operated upon eighteen patients with thyroid cancer and one of these died. This patient stayed in the hospital for many weeks after her operation until she passed away with tracheal obstruction nearly two months after the surgical procedure had been carried out.

In the groups of goitre patients which we have just considered, the surgical mortality, we have said, should be extremely low, since, with the exception of the cancer cases, these lesions are essentially benign at the time of operation and since we can, by a process of selection, avoid the more hopeless cases of malignancy. The two fatalities in this group of four hundred and twenty-two cases give a mortality of less than five-tenths of one per cent and each fatality may well be listed as in no way a contraindication to the use of surgery in this type of goitre.

Goitre surgery in the presence of hyperthyroidism is an entirely different problem than in the more benign lesions which we have been considering. Hyperthyroidism apparently acts as a definite poison when present and it must inevitably increase the risk of operative treatment. We are coming to believe after a surgical experience with several thousand patients with toxic goitre that a certain inevitable mortality is associated with the surgical treatment of this disease. In fact it would be unreasonable and contrary to general experience with the treatment of any serious disease to anticipate that any method or procedure could give constantly

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good results and no mortality. We are at times led to believe that many physicians do not appreciate the serious nature of hyperthyroidism when allowed to persist for months or years with incomplete or inadequate control. We believe that they may overlook the exceptionally serious consequences of uncontrolled hyperthyroidism in patients who have hearts previously damaged by rheumatic fever or other infection. There has rarely been a year, in our experience with thyroid disease, in which there have not been so many hospital patients who have died of hyperthyroidism unoperated, either with or without cardiac complications, as have died as the result of surgical treatment of the goitre. Thus in 1931 three patients died in the hospital of hyperthyroidism with cardiac complications who at no time in their hospital stay were in a condition when surgery could be considered even by those of us who recognize the value of radical surgery in these seemingly hopeless patients.

In considering the mortality associated with the surgery of hyperthyroidism we divided our patients into two groups—the toxic adenomatous goitre patients and the exophthalmic goitre patients. This is for the purpose of statistical information and for comparison with similar groups of patients reported by others. We performed in 1931 a hundred and twenty-seven operations on ninety-nine patients with toxic adenomatous goitres. Three of these patients died, giving a mortality of 3.03% by patients and of 2.36% by operations. We performed six hundred and forty-eight operations on four hundred and seventy-two patients with primary hyperthyroidism. Three patients died, giving a mortality by patients of .63% and by operation .46%. In view of the fact, however, that hyperthyroidism is the factor which marks out this entire group of patients from all other goitre cases, we feel that a consideration of the fatalities should include at once both toxic adenomatous goitres and exophthalmic goitre patients. To summarize the entire group of toxic cases operated in 1931, then, we find that 775 operations were performed on 571 patients with six deaths—a case mortality of 1.05% and an operative mortality of .77%.

In 1930 the author made a careful study of the mortality factors in hyperthyroidism in all fatal cases for the five years previous to 1930*. In this study I pointed out that over half of our mortality in toxic thyroid patients for five successive years was due to postoperative thyroid storms and their complications. Furthermore it was noted in this study that in each patient who died, definite information was at hand which, had it been correctly interpreted, might well have led the surgeon to do a less extensive operation than he had undertaken. It was found that the significant factors in these fatal cases in which surgical judgment played an important part were the age of the patients, practically all of whom were at middle life or beyond; the history of marked weight loss, and finally the existence of the hyperthyroidism for more than a year. Keeping these facts clearly in mind we have attempted in the past year to be more conservative with patients who fall into this class of serious risks. Yet when we analyze the fatal cases in 1931 we find again that in those patients whose deaths may be related to faulty judgment, definite information of the seriousness of the condition and of the danger of a possible fatality was present when considered with the above points in mind.

The six deaths in the operations for toxic goitre in 1931 can be quickly analyzed. They may be divided first into two groups—one, those patients in whom death was due to an accident or to a complication beyond our control and, two, those patients in whom it is quite possible that the end results might have been different had our surgical judgment indicated either longer delay before operation, a smaller operation or no operation at all.

Two of the fatal cases may clearly be classed as deaths due to accidents and not related to surgical judgment. The first patient a woman, No. 19749, aged fifty-eight, had an adenomatous goitre present for ten years and with toxic symptoms for over two years before admission. Iodine had been given intermittently in this interval. She had marked arteriosclerosis with hypertension and auricular fibrillation. Her basal metabolic rate on admission was +54. A subtotal thyroidectomy was done in two stages with a six weeks' interval between operations. Seven days after the final operation when the patient had made a most satisfactory recovery she was leaving the hospital to return to her home. She slipped and fell down the stairs, sustaining a fractured skull from which she died within a few days.

The second case in this small group was another woman, No. 20184, aged sixty-six, who had had an adenomatous goitre for four years with hyperthyroidism in the past year. Her basal metabolic rate on entrance was +36 which had dropped to +17 a week later, before operation. A subtotal thyroidectomy was done without particular difficulty or technical trials and she returned to her room in excellent condition. Some four hours later she had a sudden choking attack with hemorrhage deep in the wound and in spite of reopening of the wound, artificial respiration and stimulation, she died. It may not be out of place at this point to comment on the fact that in over ten thousand goitre operations which we have performed in the Clinic this is the first case which we have ever lost from

*The Operative Mortality in Hyperthyroidism by Howard M. Clute, *Journal of the American Medical Association*, August 9, 1930, Volume 95, pages 389-392.

hemorrhage. We have several times had very serious hemorrhages from either the superior or inferior thyroid arteries a few hours or even a day or more after operation, but they have been successfully controlled. These accidents are dangerous, not only because of the loss of blood but more particularly because if the hemorrhage is beneath the prethyroid muscles enough pressure rapidly forms to obstruct the trachea and produce suffocation. At least twice in our experience we have been able to cut the sutures holding the prethyroid muscles, release the pressure on the trachea and then, when respiration was freely reëstablished, proceed with the control of the bleeding. In this case, however, although these procedures were carried out within fifteen or twenty minutes of the first symptoms, resuscitation was impossible.

Two of the toxic thyroid deaths in 1931 occurred in thyrocardiacs. The first was a man, aged forty-eight, No. 25697, who had exophthalmic goitre probably of several years' duration. He had lost thirty-five pounds in weight and had taken iodine intermittently. He was known to have angina pectoris from his clinical story, but electrocardiograms showed no abnormality of the heart function before operation. His basal metabolic rate at the time of operation had fallen to plus three and his general condition was quite satisfactory. Operation was undertaken with the full knowledge by the surgeons and of the family that the well-known dangers of angina were present. When the right lobe of the thyroid had been removed with no technical difficulties, the patient died. All attempts at resuscitation with intravenous and intracardiac injections of adrenalin and cardiac massage were without results. Autopsy showed a thrombosis of both branches of the coronary artery.

The second patient in this class was a woman, aged seventy, No. 22644. She had had a goitre for thirty years with toxicity present for some time. She entered the hospital with congestive heart failure and in addition to her hyperthyroidism showed an old rheumatic heart disease. Her basal metabolic rate was +33 and +27. After more than a month of preparation, when the metabolic rate had dropped to +9, operation was undertaken and a large, right-sided, substernal adenomatous goitre was removed without difficulty. Five days after operation the patient died a cardiac death with, in addition, a probable renal artery embolus. These two thyrocardiacs who died were recognized as serious risks when the operation was undertaken and operation was only undertaken, particularly in the elderly woman, as a procedure of last resort. Our experience with thyrocardiacs has made us most enthusiastic about the removal of hyperthyroidism from patients whose hearts are damaged by intrinsic cardiac disease. While the mortality in these seemingly hopeless, old and feeble patients is somewhat high, we still feel convinced that the restoration of competent cardiac function in over ninety per cent of the thyrocardiacs on whom we operate, well warrants the increased risk which operation carries in this group of patients. It is certain, however, that we must consider thyroidectomy even in thyrocardiacs with great conservativeness if they are over seventy years of age.

Two patients died in 1931 with typical thyroid storms following operation for exophthalmic goitre. The first case, a woman of fifty-one years, No. 21516, had had severe hyperthyroidism for at least one year and had lost over seventy-five pounds in weight. In addition she had taken iodine for at least six months before entrance to the hospital. Her basal metabolic rate on admission was +58 and her body weight 127. Eight days later her metabolic rate had risen to 72 and her weight had dropped to 114. Eighteen days after entrance to the hospital we ligated one superior thyroid artery as a test procedure of her ability to withstand thyroid surgery. Recovery from this lesser operation was entirely uneventful and no reaction followed. Three days later, therefore, the right lobe of the thyroid was resected. The procedure was technically simple and occupied very little time. Three days after this operation, however, the patient died in a typical, severe thyroid storm.

The second case was another woman forty-five years of age, No. 22947, who had had severe exophthalmic goitre for at least two years before entrance and had taken iodine and digitalis for some time previous to hospitalization. She had lost at least sixty-five pounds in two years. She had hypertensive heart disease and auricular fibrillation. Her basal metabolic rate on admission was +83 and her body weight 149 pounds. One superior pole was tied with slight postoperative reaction. Ten days after this procedure her metabolic rate had risen to +86 and her weight had dropped to 138 pounds. Three days later the left superior pole was ligated with slight postoperative reaction. A week later on her discharge from the hospital her metabolic rate had risen to +90 and her weight had dropped to 134 pounds. She was sent home to rest for a month and on her return after this interval her metabolism was still +69 and her weight had dropped to 130. The right lobe of the thyroid was resected with relatively little difficulty. Three days later the patient died from a serious and typical postoperative thyroid storm. As we review the histories of these two patients at this time it is apparent that all the criteria which would indicate the serious nature of their disease and to which we have repeatedly called attention were present. Each patient was beyond middle

life; each patient had had hyperthyroidism for more than a year; each patient had had a tremendous weight loss and each patient had a very markedly elevated metabolic rate which failed to show any particular improvement with preliminary measures of treatment. In the first case we feel that our judgment was at fault in doing a hemithyroidectomy three days after the ligation of a single pole. It would have been much wiser to have ligated the other pole and sent the patient home for six weeks, as was our custom so constantly before the days of Lugol's medication. In the second patient both poles were ligated and an interval of four weeks elapsed before part of the thyroid was removed. In this case, however, it was evident from the constantly falling body weight and the persistently high metabolism that the disease was in a state of increasing severity. Here the fatality might have been avoided had we delayed operation until her toxicity had become less severe.

We have repeatedly emphasized the fact that multiple stage operations are necessary in the surgery of hyperthyroidism. It was our experience with thyroid disease in the days before Lugol's solution that the more conservative we were with toxic patients, the lower the mortality. We are apparently returning in some little degree to a situation similar to the pre-Lugol era in the treatment of thyroid disease. It is recognized that Lugol's solution produces in the majority of patients a marked improvement in hyperthyroidism. It is also known that this improvement is temporary and that after a time the gland becomes iodine-fast and no further clinical improvement follows the use of iodine. Certainly each of these patients demonstrates that fact and many other cases in our experience corroborate it. The general use by physicians of Lugol's solution in hyperthyroidism before recommending surgery necessarily takes the surgical treatment of thyroid disease back to a condition almost similar to the pre-Lugol era. We have a distinct impression that when patients have taken iodine for a long time before coming for surgery they are prone to have serious reaction and that their salvation depends, as in the pre-Lugol era, upon multiple stage operations. We are certain that serious or fatal thyroid reactions may occur even though Lugol's solution has been given for many weeks. The interval between the multiple stage operations in these over-iodinized patients must be made as great as it was in the days before iodine was used. Rarely, before 1924, did we ligate the superior thyroid artery and then do a hemithyroidectomy in any less than four or six weeks. This gave us good results. In seriously toxic exophthalmic goitres who have not had iodine treatment before coming for thyroidectomy rarely, if ever, will it be necessary to do a pole ligation. The use of Lugol's solution before operation, provided surgery can be undertaken when the period of improvement is at hand, will do away, we believe, with the necessity for pole ligation in a great majority of patients. Divided operations, however, are still essential for a low mortality rate in hyperthyroidism and in 1931 thirty per cent of our toxic goitres were operated upon in multiple stages. When, however, the patient comes to a surgeon for thyroidectomy, after having taken iodine for six months or a year or more, pole ligation may still well be the procedure of choice. Thus in 1931 we ligated the poles eleven times before attempting the removal of thyroid tissue and it is our feeling at present that in these over-iodinized patients pole ligation should be more frequently undertaken in the future.

From this observation of the past year's thyroid surgery it appears that accidental deaths will occur in spite of every safeguard and care in thyroid surgery. We believe that the preventable deaths in hyperthyroidism, however, will constantly be less in number because of our increasing and repeatedly stimulated interest in the danger signals of the disease and our adoption of more conservative procedures in sick patients. Long-standing toxicity, marked emaciation or great weight loss, an age beyond middle life and very possibly the long administration of iodine are the significant danger signals in these patients. Proper consideration of these factors by the surgeon; the performance of multiple stage operations with definite intervals between stages; and the avoidance of surgery in acute exacerbations of the disease should, we believe, definitely reduce the already low number of fatalities and render surgery more than ever the treatment of choice in hyperthyroidism.

THE REASONS FOR UNSATISFACTORY RESULTS IN THE TREATMENT OF CALCULI OF THE UPPER URINARY TRACT*

BY EDWARD L. PEIRSON, JR., M.D.†

THE treatment of calculi of the upper urinary tract should, theoretically, be eminently successful as such calculi usually give timely warning of their presence, and we have at our command satisfactory methods of diagnosis and practical means of treatment. In an attempt to determine how successful our treatment of this condition actually is, we have reviewed the cases of renal and ureteral stones admitted to the Massachusetts General Hospi-

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tal during the ten-year period from 1920 to 1929 inclusive. It seems to us fair to assume in this connection that the criterion of success is the removal of the stone with the minimum of kidney damage. If the stones are incompletely removed, or if, as a result of the stone or of the attempt to remove it, the kidney is seriously damaged, we must consider the case relatively unsatisfactory. Many of these factors are, however, difficult to evaluate from the records and we have therefore accepted two much broader criteria of failure: death and the necessity for nephrectomy. When nephrectomy is necessary the result must, we believe, be classified as unsatisfactory, for although many patients enjoy good health after nephrectomy, their position is not a desirable one. As renal calculi tend to be bilateral in from 10% to 20% of cases the chance that a patient who has had one kidney removed for renal calculi will develop calculi in the remaining kidney is not inconsiderable. This percentage is reported by Braasch and Foulds to be 2.75%. Likewise insurance companies' figures show that the expectancy for life is definitely decreased by the removal of one kidney.

The problems of renal and ureteral calculi are, of course, not identical. However, as most ureteral stones originate primarily as renal calculi, we have chosen to group the two conditions in a single series. A stone in the ureter may be a much more serious matter than one in the kidney because in the former position obstruction with its accompanying dilatation and infection develops much more readily. Whereas a stone may remain in the kidney for a long time without producing either serious symptoms or kidney damage, a ureteral stone sooner or later produces at least some destruction of the kidney.

Considering only the operated cases in this series, we find 190 cases that had conservative operations and 74 cases that had to have nephrectomy. That 74 cases, or 29% of the operative cases, required nephrectomy shows, we believe, that the treatment of urinary calculi is not so efficient as should be expected. Furthermore, 24 or nearly five per cent of all cases of calculi of the ureter or kidney admitted to the hospital during this period, died. Eight of these had no operation during their last stay in the hospital but two of the eight had had a nephrectomy done less than four months previously. As these two cases died of renal insufficiency the nephrectomy must have contributed to their death and probably if all the cases had been followed, we would have found other cases dying not long after nephrectomy. Six cases died following nephrectomy. Of these one died of cancer of the ovary and one of tuberculosis. Excluding these two cases we find the mortality for nephrectomy to have been five per cent. One case died of peritonitis from the intraperitoneal rupture of a perinephritic abscess which resulted from a calculus pyonephrosis. Nine cases died following conservative operations on the kidney but of this number three had had a previous nephrectomy and three were in an extreme state of uremia and the operation was in these cases only a desperate attempt to secure kidney drainage. Thus we have left three fatal cases following elective operations for the removal of stones. This makes the mortality for the elective removal of stone from the upper urinary tract one and five-tenths per cent. These figures are comparable with those reported by the Mayo Clinic and prove that conservative renal surgery is a relatively safe and satisfactory procedure if the patient is in fair condition.

An analysis of these unsatisfactory cases shows several factors which contributed to these relatively poor results. The problems of recurrent stones, of stones incompletely removed at operation, and of infection in the kidney inadequately treated after the removal of the stones are very important if renal calculi are to be treated satisfactorily. Fourteen per cent of the cases requiring nephrectomy had previously had stones removed surgically from the same side. In these cases the recurrence of stones and particularly the failure to clear up the existing renal infection were of major importance as causes of failure. However, as these factors accounted for only a small percentage of the failures and have repeatedly been discussed in the literature we shall turn our attention to what we found in this series to be the chief cause of failure in the majority of cases: *Delay*. Considering the 74 cases which came to nephrectomy, we find that pain was the first symptom in 63 cases; 42 cases had complained of pain for a year or more and 14 had complained of pain for over 5 years. Furthermore, 58% had complained of severe pain which was more or less characteristic of calculi in the kidney or ureter for an average duration of at least 29 months. Knowing how rapidly a kidney may be destroyed, it seems quite justifiable to assume that, in these cases at least, the treatment could have been started at an early time when complete conservation of renal tissue would have been possible. In addition 15 of the 74 cases of nephrectomy gave a history of gross hematuria over an average period of 12 months. The admission specimen of urine showed albumen in 45 cases and red cells in 20. Seven had an N.P.N. on admission over 40 and 14 had a two hour renal function under 35 per cent. In regard to other complications we find that in six cases perinephritic abscess developed as a result of renal calculi and two other cases had carcinoma of the pelvis of the kidney. Although only a very small number of cases of renal calculi develop

cancer of the pelvis, the figures of Miller and Herbst, who found stones associated with malignancy in over twenty per cent of 54 cases of papillary epithelioma of the renal pelvis, show that there exists some degree of causal relationship.

These few figures give some concrete evidence of the amount of time which was wasted before these patients actually came to operation, and show why so many cases required nephrectomy but it is impossible to present with figures a picture of the golden opportunities which were lost and the unfortunate consequences which followed in many of these cases as a result of delay. This delay seems to have been due to three causes. In the first place it was due to indifference on the part of the patient, who feeling relatively well between attacks of severe pain refused to enter the hospital for treatment. The medical profession has been very intent on teaching the public the necessity for treating cancer early, but has allowed them to consider renal calculi as relatively unimportant. Yet even early treatment of cancer is often unsuccessful whereas the early treatment of renal calculi usually produces brilliant and lasting results. In the second place, the delay has been due to the family physician who either failed to make the diagnosis or continued too long with expectant treatment in the vain hope that the stone would pass of its own accord. Thirdly, this disastrous delay has been due in some cases to the urologist who has persisted too long in his attempts to remove the calculi by non-operative procedures.

In this respect we wish it clearly understood that although we consider the non-operative treatment of ureteral calculi far from being free from danger, we do not in any sense advise operation on all cases of calculi of the upper urinary tract. We realize very clearly that many ureteral stones will pass or can be caused to pass without operation, and that some cases of renal calculi are better left alone. This is obviously the case where the removal of a calculus will cause more damage than is being caused by its presence in the kidney. We do feel, however, that every case of calculus should be carefully studied, carefully followed, and treated according to the requirements of the individual case. There are many reports in the literature about the non-operative removal of ureteral stone. Crowell reports ninety-two per cent and Ballenger and Elder report ninety-five per cent success. Other surgeons have been successful in a smaller percentage of cases, Beer and Hahn reporting sixty per cent and Barney and Chute reporting fifty-one per cent successfully removed by cystoscopic manipulations. Ureteral stones can be treated safely by cystoscopic manipulations only when they are small enough not to completely obstruct the flow of urine and when there is no infection present. If infection or signs of increasing kidney damage develop, operative removal should, we believe, be carried out at once. If no progress has been made in removing the stone after two cystoscopic attempts, it seems to us to be unwise to continue with this form of treatment. The operative mortality for conservative operations on the kidney is usually reported as less than two per cent, while Judd reported 400 ureterotomies with but a single death. On the other

PLATE IA.

A pyelogram done when this patient (Case I) was first seen, showing marked dilatation of the pelvis, calices, and ureter caused by a small stone in the lower ureter.

hand Bumpus and Thompson point out very clearly the dangers of attempting to remove stones from the ureter by cystoscopic manipulations. In 274 cases in which this was attempted by them, suppurative pyelonephritis developed 32 times (eleven per cent) and two patients who refused operation after this complication had developed died. Likewise, in the management of stone in solitary kidney or ureter Mathé considers early operation the method of choice as in such cases the treatment must of necessity be sure and efficient.

The dangers of continuing too long with the expectant and non-operative treatment in cases of renal and ureteral stone can perhaps be more clearly shown by discussing briefly six cases not

included in this series. These cases which are in no sense remarkable or unusual have been chosen because they appear to us to be ordinary cases of calculi which illustrate very clearly, each in a different way, the topic of this discussion. The first case, a man of 47 (Salem Hospital 5233), is presented simply to emphasize the remarkable powers of regeneration possessed by the kidney. Plate IA shows a pyelogram done when the patient was first seen. At this time there was marked dilatation of the

PLATE IB.

A second pyelogram of the kidney shown in Plate IA done one year after removal of the stone. This shows how completely nature has been able to repair the damage caused by an obstructing stone if it is removed in time.

pelvis and ureters due to the presence of a small stone in the lower end of the ureter. This was removed by cystoscopic manipulation. A year later (Plate IB) a second pyelogram was made which shows how completely a kidney will return to normal if an obstructing calculus is removed in time. Unfortunately, however, the stones are not always removed in time and too often we see such distressing cases as the second case (Salem Hospital 52308, Plate II). This case was an intelligent American woman of 50 who had had frequent, severe attacks of renal colic over a period of 8 years. The diagnosis of renal calculi had been made by her family doctor and she had been advised to have something done; an excellent piece of advice which was not followed. She was seen by us only after she was in an extreme state of uremia and anuria. At this time the N. P. N. was 100 and the case seemed hopeless. Following immediately upon a cystoscopy which failed to relieve the anuria, a left nephrotomy with removal of the large stone was done. She stood the operation well and immediately afterward began to drain urine from both kidneys, proving that the anuria was of a reflex nature. Unfortunately, one week after operation she developed severe sepsis in the other kidney. Nineteen days after the first operation the N. P. N.

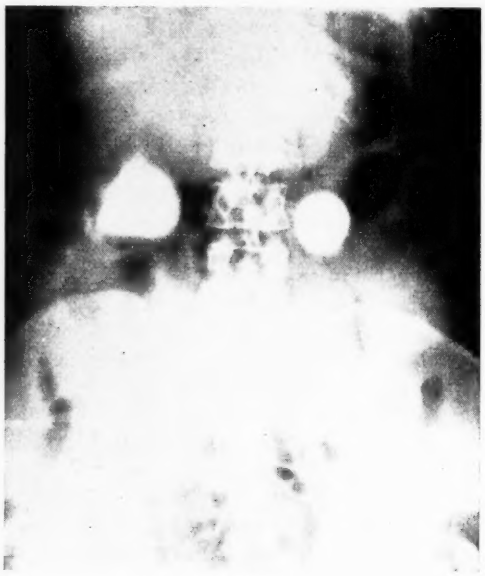

PLATE II.

A plain x-ray of a hopeless, neglected case of bilateral renal calculi. Case II.

had dropped to normal but the sepsis in the right kidney demanded drainage which was carried out in spite of the patient's very poor condition. The right kidney was found to be the seat of a severe and diffuse pyelonephritis. The stones were removed and the kidney drained but the patient did not have sufficient strength to stand the operation and died the following day. It seems almost impossible that anyone could be so foolish as to delay treatment as long as was done in this case, yet we have recently seen a similar though less advanced case in a clever and well-educated lawyer, who in spite of all we could say has refused to have even a cystoscopy performed. Obviously, the only method of preventing such disastrous results is through education of the public, which is in the last analysis the duty and the privilege of the medical profession.

The third case (Salem Hospital 53440) illustrates the dangers of attempting to remove a ureteral calculus by cystoscopic manipulations. We were able, in this case, to demonstrate by means of a wax-tipped catheter a small non-

opaque ureteral calculus in the left ureter. This man was 67 years old and his general condition was not good. As the stone was not large enough to obstruct the passage of the catheter and as we did not know in exactly which part of the ureter it was situated, we persisted in spite of kidney infection, which was present at the start, in trying to remove the stone by non-operative procedures. The N. P. N., which was 50 when we first saw him, rose rapidly until

PLATE IIIA.

A pyelogram taken when the patient, Case III, was first seen with a small stone in the lower ureter.

two weeks later it was 80. In spite of the use of inlying ureteral catheters, his condition rapidly became so bad that operation became impossible. Fortunately, during the fourth cystoscopy two weeks after the first, we were able to remove a very small calculus from the left ureter. Following this he made a long but satisfactory recovery. The pyelogram in Plate IIIA shows the condition as it existed when we first saw this patient. If this is compared with Plate IIIB taken 17 days later, one sees very clearly how rapidly the presence of even a very small stone in the ureter will cause destruction of a kidney if infection is also present. Undoubtedly, in this case operation, even in spite of the patient's poor condition, would have been a safer and more satisfactory procedure. A pyelogram taken three months later showed the return of the kidney to nearly normal, but the pus has persisted in the urine.

The fourth case likewise shows how rapidly a kidney can be destroyed where infection and obstruction coexist. This case, Salem Hospital No. 54531, was a young girl of 22 who had never had any sickness of any kind. While visiting friends in the country she suddenly developed severe pain in the left costovertebral region associated with fever and vomiting. She was treated at home with urinary antiseptics and fluids but as her condition did not improve she was sent into the hospital. Cystoscopy revealed a stone 1 cm. in diameter in the upper end of the

PLATE IIIB.

This pyelogram is of the same kidney shown in IIIA taken 17 days later. It shows extensive destruction of the kidney due more to infection than to obstruction and proves that an attempt to remove stones by cystoscopic manipulation is not without danger.

PLATE IIIC.

Pyelogram of the same case as IIIB taken three months later, showing repair of the kidney damage.

ureter (Plate IV). Operating on the following day (only nine days after the first symptom) we found a small stone impacted in the upper ureter, a large pyonephrotic kidney with a cortical abscess involving the middle third and a perinephritic abscess. Obviously, there was

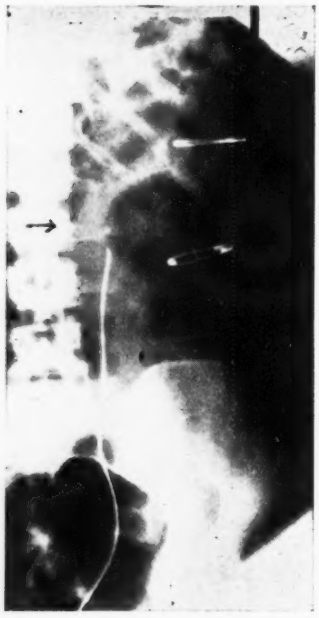

PLATE IV.

This x-ray of Case IV shows a stone which caused complete destruction of the kidney in nine days after it moved from the kidney pelvis into the ureter.

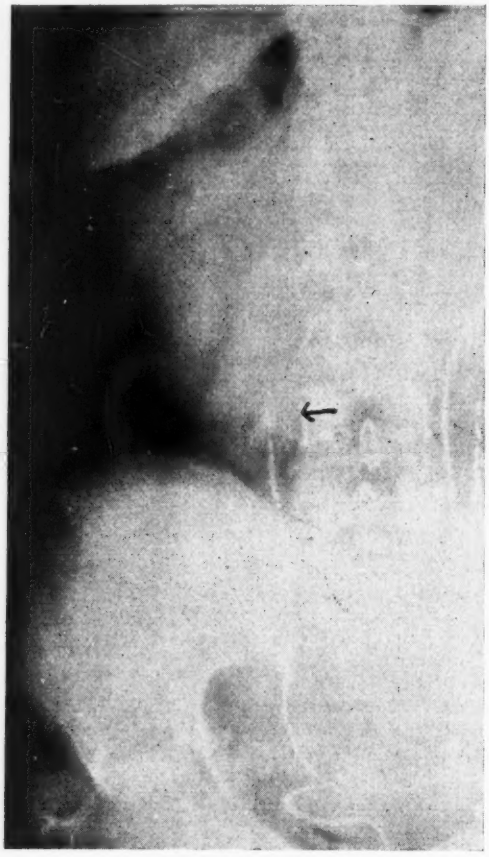

PLATE V.

An x-ray of Case V showing a small stone in the upper ureter which obstructs the passage of the catheter.

nothing which could be done except a nephrectomy. However, if the expectant treatment had not been persisted in and the operation had been performed early, it would in all probability have been an easy matter to have removed the stone and saved the kidney.

The fifth case, Salem Hospital 53998, shows how the destruction of kidney tissue may progress without presenting marked clinical signs. It is important to note in this connection that not an inconsiderable number of cases of calculus may show a normal urine, and that accordingly, a negative urine examination does not rule out this diagnosis. In our series of 74 cases which showed such severe kidney damage that nephrectomy was necessary, 22 showed no albumin, red cells or white cells in the urine

PLATE VI.

A plain x-ray (not a pyelogram) of Case VI which shows a huge silent stone filling the left kidney pelvis and three small silent stones blocking the lower end of the right ureter.

at the first examination. This case was a man of 65 who was perfectly well until two weeks before admission when he had a severe attack of pain in the left lower quadrant which lasted one day. Cystoscopic examination showed a small stone impacted in the upper end of the ureter (Plate V). Over a period of three weeks two attempts were made to dislodge the stone by cystoscopic manipulations. During this period he was carefully followed and showed no signs or symptoms of any kind. The urine analysis was repeatedly negative but the two hour renal function steadily decreased from thirty-five per cent to thirty per cent to twenty-five per cent. At this point the stone was removed surgically, and two weeks later the renal function had risen to sixty per cent.

The sixth case, Salem Hospital 54818, is a case of bilateral silent stones which shows that the absence of pain by no means excludes the diagnosis of renal or ureteral calculus. The only symptoms

shown by this patient (a man of 36) were vomiting and loss of strength, but examination of the urine sediment showed pus. In the course of a G. I. series a large stone filling the entire pelvis of the left kidney and three small stones in the lower end of the right ureter were found (Plate VI). Further study showed that his symptoms were uremic in origin. Believing that the left kidney was destroyed beyond hope of repair it was thought that the stones should be removed from the right ureter at once without jeopardizing the right kidney in any way. Accordingly, the case was considered primarily an operative one and no attempt was made to remove these stones by cystoscopic manipulations. A right ureterotomy was performed at once and the patient made an uneventful recovery and has remained in excellent health.

SUMMARY

An analysis of the cases of ureteral and renal calculi admitted to the Massachusetts General Hospital over a ten-year period shows that the results obtained in the treatment of these cases are not so good as should be expected. Further analysis of these cases shows that the greatest cause of failure is due to delay in performing the operation. It seems desirable that the public and the medical profession realize more clearly the seriousness of urinary calculi. We believe that every patient who is suspected of having a urinary calculus should have both a complete urine analysis and an x-ray. If a stone is discovered the patient should be cystoscoped and carefully followed until the stone has been removed. If attempts are made to remove the stone by non-operative measures the case must be very carefully followed from every angle and operation performed upon the first appearance of signs of infection or increasing kidney damage. Expectant treatment can be safely carried out only after a complete urological examination and in cases where the patient will be continually under rigid medical observation.

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A NEW METHOD OF TREATING THE INDIRECT SAC IN INGUINAL HERNIORRHAPHY

BY EDWARD M. HODGKINS, M.D.*

THE proper treatment of the indirect sac in inguinal hernia is of very first importance in the cure of this condition. No amount of narrowing of the external ring, obliteration of the canal or plastic work on the external oblique aponeurosis will prevent the reformation of a sac if the entire peritoneal protrusion is not completely obliterated. The commonly practiced method of simple amputation high on the neck of the sac, allowing the stump to recede freely and without fixation of some sort, is unquestionably responsible for an indeterminate percentage of recurrences. Security in this method is entirely dependent on the strength of the catgut and no additional barrier. Postoperative strain from coughing and vomiting has a definite weakening effect on the stump which is the most vulnerable point in the whole repair. Sepsis is even more disastrous.

The Kocher method of dislocating the amputated stump of sac beneath the edge of the internal oblique muscle is a classical procedure which for years has enjoyed a preference over all others. Macewen's operation on the sac has also been very popular and provides for an infolding and plication of the entire unopened sac with dislocation upward through a channel between the peritoneum and internal oblique muscle. Both of these methods have been practiced by those who believe that something more than simple amputation adds to the security of the repair, and they still serve the purpose admirably.

A few surgeons have called attention to the fact that some of the sacs, especially the early ones, are so thin, filmy, short, et cetera, that it is not possible to do these fixation operations upon them. This has not been my experience.

I have personally done the Kocher and Macewen operations a sufficient number of times and under such varying conditions with regard to the consistency of the sac, to feel sure that in the vast majority of cases they can be used. Furthermore, the treatment of the indirect sac as described and illustrated in this paper has been

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carried out routinely and without exception in more than 300 cases although it is even more dependent on the well-formed sac than other methods.

Many surgeons are now becoming convinced of the futility of placing any type of constricting suture in the muscle. That such treatment causes atrophy and retraction of muscle with consequent loss in efficiency is an established fact. In primary hernia, the well-developed and intact muscle should be left entirely undisturbed so that it can go on performing the full normal function for which there is no substitute. If the building of a new tissue wall is demanded on account of a thin frayed-out muscle, some fascia method suited to the individual anatomy can be employed. The preservation of muscle under any condition is most desirable.

We also find an increasingly larger group of herniotomists coming to the belief that in the majority of adults with firm muscular walls and in all children, proper treatment of the sac alone is sufficient to effect a cure. They are giving up all plastic repair of tendon and fascia layers, recommending only simple anatomical closure. R. H. Russell of Australia has been the leader in this thought and we find that since this author's first publication on the subject, he has won many adherents. The literature is abounding in comment from other authors indicating that they now realize that he is at least in a certain measure correct.

Although I am still of the opinion that some type of fascia plastic adds to the security of the repair, I have willingly tried simple anatomical closures in appropriate cases and shall follow and compare the results in these patients with great interest. To those operators who are of the same mind, I would suggest that the following description offers a method of handling the indirect sac more satisfactorily.

In *Surgery, Gynecology and Obstetrics*, for December, 1928, I published a paper entitled "A New Method of Inguinal Herniorrhaphy with Living Fascial Sutures Obtained from the Rectus Sheath." While the main portion of the paper dealt with the plastic repair by living sutures, I attempted at the same writing to include a brief description of an original method of treating the indirect sac. That such description was much too brief, I have since been convinced, on account of the many requests coming to me for a more detailed explanation of the technique employed.

AUTHOR'S METHOD

Exposure of the inguinal canal is completed by incising the aponeurosis of the external oblique muscle from the external ring upward. The inner leaf at the upper angle of this incision is separated from the muscle about 2.5 cm. mesially to expose the muscle.

The indirect sac is separated from cord structures, dissection being carried well up to the parietal origin of the neck, care being used not to injure or tear the sac at this point. Should such an accident occur, the rent must be repaired by suture. The sac is then held up perpendicularly, opened, inspected, and contents reduced.

At the highest point on the neck and always under direct vision, a curved needle carrying chromic No. 2 catgut is passed through the middle of the sac and tied on one side. The needle is again passed through the middle in the opposite direction to the first suture and tied on the opposite side. One or two more sutures carried back and forth just below this point firmly transfix the sac. Both ends of the catgut suture should be left long. Amputation is now done in the usual manner leaving a cut stump above the suture line about 1 cm. in height.

First Step. The left index finger is now inserted under the internal oblique muscle at the point where the fibers arch. A channel of separation between the muscle and parietal peritoneum is easily made by a twisting and burrowing motion. Fig. 1. The channel should be

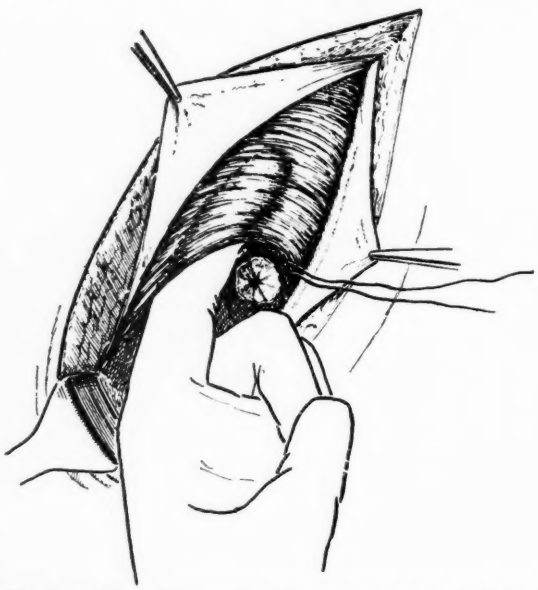

FIG. 1. Finger burrowing between muscle and peritoneum to create channel. Stump of sac prepared, with loose sutures hanging free.

narrow, and never more than the width of the finger. Wide lateral separation is unnecessary and technically incorrect. The distance of channeling can be judged by the position the stump of the sac occupies in relation to the border of the muscle. For example; when the stump is displaced upward to the new position, there should be sufficient tension exerted on the parietal peritoneum below to take up the slack in the canal. (Shown in the fourth step.)

Second Step. With probe, pointed scissors, or hemostat at a point on a level with the tip

of the finger and slightly mesial to it, an aperture is made through the muscle by separation of the fibers in the direction of their course. Tearing of the fibers is to be avoided. Fig. 2.

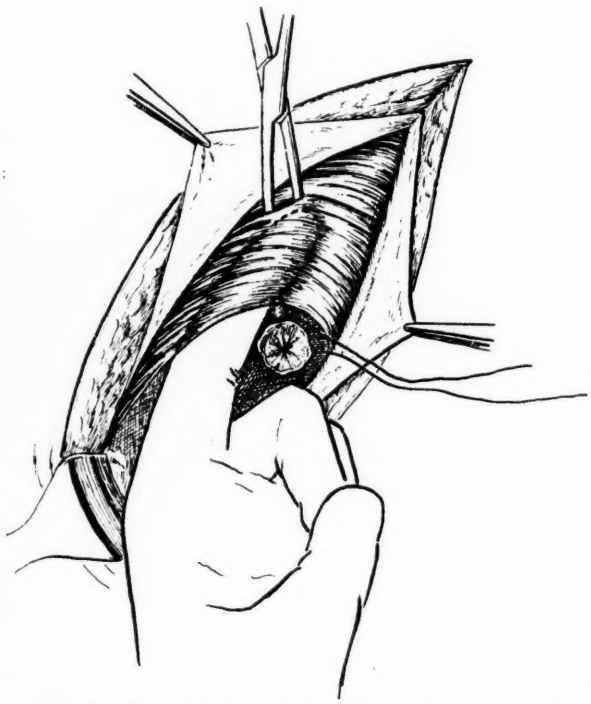

FIG. 2. Hemostat plunged through muscle to level of tip of finger and slightly mesial, making aperture down to peritoneum.

Third Step. The tip of the hemostat is pushed into the opening through the muscle to contact with the finger. The finger is then withdrawn

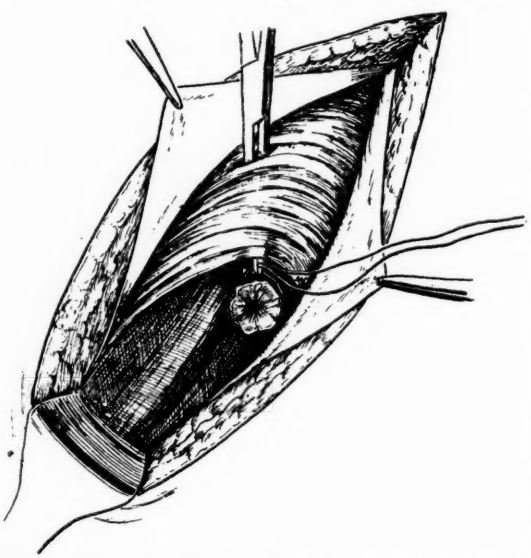

FIG. 3. Hemostat directed through channel downward as finger is withdrawn. Loose ends of suture then grasped in hemostat preparatory to drawing stump of sac upward through channel.

followed by the hemostat along the channel downward and outward between the border of the muscle and peritoneum. The loose ends of the catgut transfixion suture are then grasped close to the stump of the sac. Fig. 3.

Fourth Step. The stump of sac is then drawn upward through the channel and outward through the opening in the muscle. The two ends of suture are re-threaded on needles and carried through the medial leaf of the aponeurosis from below upward 1.5 cm. apart and tied together on the external surface. Fig. 4.

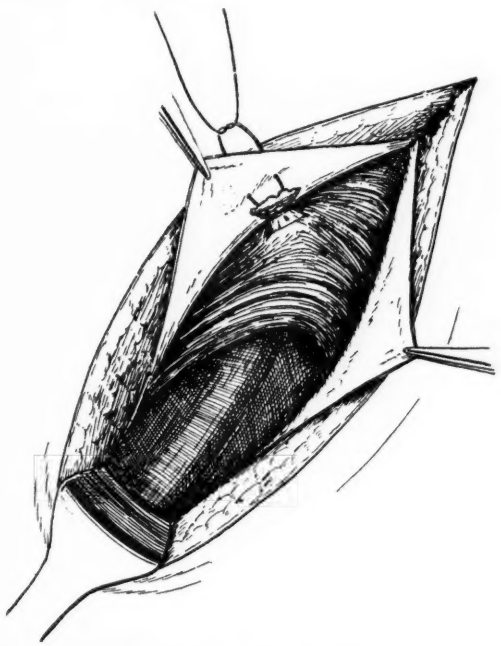

FIG. 4. Stump of sac drawn upward through channel and outward through aperture in muscle. Two ends of suture shown passing through medial leaf external oblique aponeurosis. Peritoneum in floor of canal is drawn tightly. Knot preparatory to tying externally.

ADVANTAGES

1. The force of straining in the inguinal canal is obliquely forward and downward. The method herein described provides a means of dislocating the stump of the sac away from that force of strain. This is important in the event of respiratory complications or prolonged ether vomiting directly following operation.

2. The neck of the sac has been drawn through a powerfully contracting muscle which exerts a sphincteric action under strain.

3. The fixation suture is tied on the external surface of the aponeurosis drawing the cut surface of the stump of the sac firmly into contact with fascia. This means firm anchorage by adhesions to an unyielding structure and is histologically sound.

4. Sutures are not tied over muscle (an objection frequently cited with other methods) thereby avoiding muscle atrophy and interrupted functional tone.

5. There can be no possibility of accidentally including the iliohypogastric nerve in sutures or ties.

6. Should infection occur, the stump, which is the most vulnerable point in the repair, is isolated far from the general field.

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A SMALL MILK-BORNE OUTBREAK OF SEPTIC SORE THROAT*

BY ROBERT E. ARCHIBALD, M.D.,† AND ROY F. FEEMSTER, M.D.†

INTRODUCTION

THE outbreaks of septic sore throat usually reported in the literature have been the spectacular ones involving a large number of cases. These are fortunately becoming fewer because of the rapid strides which are being made to protect milk supplies, particularly by the more common employment of pasteurization. Small outbreaks will continue to occur, of course. as long as there are unsupervised milk supplies. This report deals with such an outbreak, fortunately limited to the users of milk from a single cow. The outbreak was not brought to our attention until four days after the death of the fourth victim, who was owner of the cow. If the milk had been mixed with a large supply, another catastrophe like that in Lee, Massachusetts, in 1928 might have occurred, for the organisms were very virulent, the attack rate and the fatality rate being unusually high.

CLINICAL PICTURE

The usual clinical picture was that of a sudden onset with chilliness, fever and a dry, raw throat. There was also nausea, vomiting, backache and prostration. All those attacked were sufficiently ill to call a physician. The temperature varied with the individual, some showing a pyrexia as high as 105°. Examination of the throat showed a bright red pharynx, uvula and soft palate, no exudate being visible.

In all of the cases there was more or less swelling and tenderness of the cervical glands, this being a marked symptom in six instances. No other complications occurred except in the three fatal cases, two of which developed pneumonia, one within two days and the other within four days after the onset of sore throat. The third fatal case developed otitis media and cellulitis which began in the eyelid and spread downward over the face and neck and on to the chest, the probable cause of death being septicemia. Where there was a clear history of having taken the milk only once among these cases, the symptoms usually began from twenty-four to forty-eight hours afterward. The duration of the disease in those who recovered was from three to five days of severe symptoms and a period of several days more before the end of convalescence.

Throat cultures were taken from fifteen of the cases, all of them showing hemolytic streptococci. Several patients continued to harbor organisms in their throats until convalescence was almost complete. Cultures from three patients continued to yield hemolytic colonies even after complete clinical recovery, one continuing to be positive seven weeks after the onset. Whether these organisms were of the same strain as the one which caused the sore throat was not determined. Most of the cases, however, showed negative cultures within a few days after disappearance of acute symptoms.

DISTRIBUTION OF THE CASES

Topsfield is a town of just over 1,000 population. More than half of this number is concentrated in a village near the center of the town. Two milk dealers supply most of the milk to those who do not own cows. There were no cases of sore throat in families taking milk from these dealers except in one instance. This exception was a family that bought a quart of the infected milk through their dealer.

No cases of sore throat of any severity occurred during the outbreak except among those who used milk from the infected cow. There were rumors of other people having sore throats, but in each instance investigation revealed that they had some kind of upper respiratory infection, but that sore throat was not one of the important symptoms.

One or more individuals in each of ten families used milk from the infected supply, cases occurring in nine of them. In five families every member was ill. There was a total of 16 cases in Topsfield. The owner of the cow, a man of 87, was one of the earliest cases. Complications, including pneumonia, rapidly developed and he died within three days after the first symptom. A neighbor, a woman of 75, developed a sore throat but refused to go to bed or to take care of herself properly. She died three hours after arriving at a contagious disease hospital on the seventh day of her illness, the cause

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of death being septicemia following otitis media and cellulitis.

Four cases developed among residents of other towns, all of those affected being relatives of the owner of the cow. A grandson and his wife visited in the home of the owner of the diseased cow, drank milk and took a quart home with them to Norwood. Both had severe sore throats and were in bed two or three days. A son of the owner, his wife, and five daughters from Lynnfield were in Topsfield at the owner's funeral. Five of them used milk which they had brought with them; two of the daughters used milk from the infected cow, one having a severe case of sore throat, the other escaping. A daughter of the owner and her son from Haverhill were at the funeral. The mother drank the milk, developed a sore throat and died of a complicating pneumonia and septicemia. The son did not use the milk and was not ill.

Three or four quarts of the milk were bought by a local drug store when its supply ran short and most of it was served in milk shakes to automobilists who stopped as they passed through the town. The drug clerk who prepared the milk shakes drank some of the milk and developed a sore throat. Only three of those who drank milk shakes could be traced; none of them were affected.

One of the milk dealers frequently obtained a quart of milk from the owner of the cow when his own supply was short. Two days before the nature of the outbreak was discovered, he took two quarts and delivered them to his customers. The milk was already bottled and was not mixed with the rest of his supply. He could remember where he had delivered only one of the quarts. Two members of the family used the milk, one became ill. A third person roomed in the house, but used milk from another source and did not have a sore throat.

The distribution of the cases by families is shown in Table I. Families A to I were the residents of Topsfield who used the milk more or less regularly. The family escaping (I) the infection claimed to use the milk only in cooking. Families X, Y and Z were out-of-town relatives visiting in the home of the owner.

The total number of individuals in the families where a case occurred is shown by age groups in Table II. It will be noted that there were 13 individuals in the 13 families who claimed that they had used none of the milk. Although they were exposed to the cases in their homes, they failed to develop the disease, indicating that the disease was not easily transmissible by contact. There were three individuals who claimed to have used the milk only in cooking. Only four persons known to have used the milk raw failed to become ill during the outbreak. One of these developed the disease a week after the distribution of the milk was stopped, being apparently the only secondary case in the outbreak. Her husband had had a sore throat and was still ill at the time of her onset. Thus only three persons who used the milk raw escaped the disease. One of these (in family X) used a small quantity of milk mixed with cream from another source on cereal on one occasion only.

TABLE I

DISTRIBUTION OF CASES BY FAMILIES

Family	No. Members	No. Cases	Attack Rate	Remarks
A	3	3	100%	1 died (owner of cow).
B	2	2	100	1 was a secondary case.
C	3	3	100	
D	3	3	100	
E	2	1	50	
F	1	1	100	Died.
G	2	1	50	Used only 2 quarts during outbreak.
H	3	1	33	1 did not use milk.
I	2	0	0	Claimed to use milk only in cooking.
J	7	1	14	Drug clerk drank milk at store; others used another supply.
X	7	1	14	2 used milk only once; others not at all.
Y	2	2	100	Both used milk.
Z	2	1	50	1 died; other did not use milk.
	39	20	51%	

THE MILK SUPPLY

All the foregoing epidemiological findings pointed to the one small milk supply as the cause of the outbreak. The milk was obtained from a single cow. The owner, because of his age, hired a man to care for and milk the cow. The milk was strained and put into bottles by a 65 year old housekeeper. It was not delivered, neighbors coming to the house when they wanted milk. They either brought a container into which the milk was poured or brought an empty bottle and carried away a full one. Only three or four families took the milk regularly, the others getting a quart at irregular intervals. The fact that the housekeeper had an acute attack of

septic sore throat late in the outbreak suggests that she was a victim and not the cause of the outbreak.

The milker, a man of 70, claimed not to have been ill, and also maintained that there was nothing the matter with the cow. Examination of the cow, however, revealed that the right forequarter of the udder was caked (mastitis) and a scab covered the end of the teat, making it difficult to force milk out. When the mastitis was called to the attention of the milker, he made light of it, acknowledging that he had noticed the condition a week before and had been applying camphorated oil to the udder in hope of softening it. A throat culture taken from the milker showed numerous colonies of hemolytic streptococci, as did also the milk from the affected quarter; the other three failed to yield the same organisms.

It would appear that the cow had become infected by organisms from the throat of the milker. An inflammatory condition was set up which favored the rapid multiplication of the streptococci. When milk from the other three quarters was mixed with that from the infected quarter, the whole became heavily seeded with bacteria.

TABLE II

AGE OF PERSONS EXPOSED AND CASES OF SORE THROAT

	Persons in Families with a Case of Sore Throat				Cases	Attack Rate	
	Did not use milk	Used milk cooked	Used raw milk	Total		Among total in families	Among those using raw milk
0-4	—	—	—	—	—	—	—
5-9	1	—	—	1	—	—	—
10-14	3	—	—	3	—	—	—
15-19	3	—	3	6	2	33%	67%
20-29	—	—	2	2	2	100	100
30-39	4	—	2	6	2	33	100
40-49	2	—	5	7	4	57	80
50-59	—	3	6	9	5	56	83
60-69	—	—	2	2	2	100	100
70-79	—	—	1	1	1	100	100
80 and over	—	—	2	2	2	100	100
Total	13	3	23	39	20	51%	87%

CONTROL MEASURES

Practically all of the cases had developed before the outbreak was brought to official attention. Only two new cases developed after the investigation began, one apparently being a secondary case. The local board of health immediately ordered the sale of the milk stopped. The cow was ordered slaughtered so that there would be no chance of her getting into a dairy herd and causing a larger outbreak. Autopsy showed a diffuse mastitis from which hemolytic streptococci were recovered. The milker was prohibited from milking or handling milk until two negative throat cultures were obtained.

SUMMARY

Twenty cases of septic sore throat were definitely traced to milk from a single cow. The attack rate was high, 87% of the persons definitely known to have used the milk raw becoming ill. There were three deaths, giving a fatality rate of 15%. Hemolytic streptococci were found in cultures taken from the throats of fifteen of the cases and from the throat of the milker, and in milk from the diseased quarter of the cow's udder.

THE CAUSATION AND PROPAGATION OF THE HEART BEAT*

BY WILLIAM D. REID, M.D.†

WHY does the heart beat? Most of the physiologists whose writings I have consulted content themselves with a statement of the fact that the specialized tissue of the heart possesses the power of initiating an impulse to contract, and they then pass on to a discussion of the various phenomena associated with the progress of the beat over the heart. It is well-known that the heart beat, as well as the contraction of non-cardiac muscles, is accompanied by an electrical wave, or in other words by a change in electrical potential. But this statement does not inform us of the mechanism that initiates the process.

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Nevertheless, by reading the literature on biochemistry and physiology one gleans facts here and suggestions there which when brought together form, in my opinion, a reasonable explanation of the origin of the heart beat. It is the purpose of this article to offer an explanation so derived.

BIOCHEMICAL DATA

The chief constituents of cells,—in fact nearly all the primary constituents except the inorganic salts,—are organic colloids, and therefore the properties of cells are largely dependent upon the properties of the colloids*.

In another group of substances, known as the crystalloids, the molecules tend to divide into two or more parts which have different electrical charges. These electrically charged particles are known as ions. The ions of crystalloid substances, especially inorganic salts, tend to affect colloids by the action of their electrical charges.

An important character of colloids is the possession of electrical charges on the particles of which they are composed. A protein molecule in a slightly alkaline solution acts as a weak acid, and similarly tends to send off positively charged hydrogen ions into the solution. On the other hand, in an acid solution a protein acts as a base—resembling in this way the amphoteric amino-acids of which it is composed—and so tends to split off negatively charged hydroxyl ions so that the remaining bulk of the molecule forms a positively charged ion‡.

According to Lillie[3], two of the most fundamental properties of living matter are its electrical sensitivity, and its production of electrical currents during activity†. This worker builds on the "membrane theory" of Bernstein[4] who considered the protoplasm to be separated from the surrounding medium by a semi-permeable membrane, which permits the passage of ions at varying rates. This factor of semi-permeability causes ions which are being transported to and from the protoplasm to collect in greater concentration on the thin protoplasmic surface film. The latter by means of the electrical charges carried by the ions (the particles into which the molecules of substances in the crystalloid state tend to divide) acquire an electrical potential.

Lillie[3] asserts that electrical stimulation is the primary form of stimulation. The flow of ions to and from the semi-permeable surfaces of the irritable tissue produces change in the electrical surface polarization, and this is the primary condition of stimulation. He states: "On the basis of the law of polar stimulation we may now say further that a change of polarization in a definite *direction*, such as to render the external layer of the solution in contact with the cell surface *less positive* than before, i.e., a *depolarization*, is the critical or initiatory event in stimulation."

Osterhout, in a recent Harvey Lecture[5] gives much information which seemingly applies to the causation of the heart beat. He reports experiments conducted on Nitella, a fresh water plant with cells six inches or more in length*. This authority states that there are differences in the electrical potential in the protoplasm and surrounding fluids and that this potential difference depends upon a local difference in the concentration of the ions. There tends to be an interchange of ions between the colloid of the cell and the surrounding fluid. In the cell of Nitella, whenever the electrical potential of a part could be made more negative, as was induced by the application of dilute solutions of potassium chloride, it became negative to the remainder of the cell, and, in agreement with a law of electricity, an electrical current was induced. This current flows in the galvanometer from the resting part to that in which the potential differences have become negative, but, as Wright[6] reminds us, in the cells or tissue it goes the reverse way (see Fig. 1); in other words, the part becomes excited when some or all of its potential difference has disappeared†.

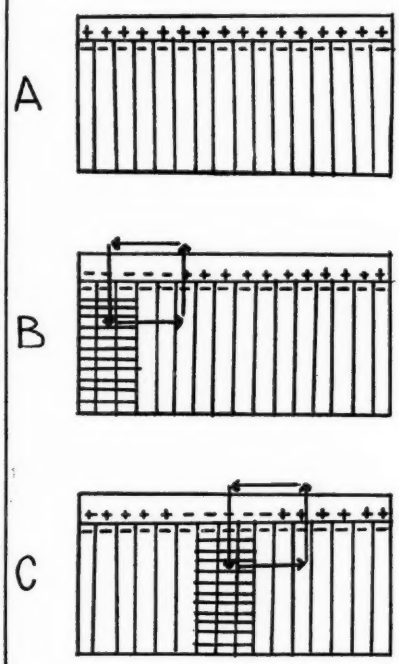

FIG. 1. Diagram of flow of electrical current in a cell of cardiac muscle. A=resting phase; B and C=active phases. In B, the active state is represented in the left end of the cell. In C, the active state has moved toward the right. The arrows indicate direction of current. The + and — signs indicate the sign of electrical potential at the cell membrane.

The process of stimulation appears to be inseparable from that of transmission or conduction. The effects of local alteration in protoplasm tend to spread, i.e., to produce chemical and physiological effects in regions other than

*Ref. 1, p. 32.
†Ref. 2, p. 245.
‡Pp. 104, 269, 274 and 283.

*P. 169.
†P. 184.

those immediately acted upon by the stimulating agent*. The essential feature of the process of transmission is the breakdown and reconstruction of the thin protoplasmic surface-film under the influence of the local bio-electric circuits*. The local circuit arising between this electrically altered region and the as yet unaltered regions adjoining forms the next link in the chain of events; and if the local current has sufficient intensity and local density to break down electrolytically the film over a certain area of the adjoining region, an indefinite wave of propagation may be initiated, since the same effect will be repeated at each newly formed boundary between the active and inactive area.

The phenomena pertaining to an impulse in a nerve are strikingly similar to those occurring in the cardiac tissues. According to Gerard[7], "The impulse has been defined as a propagated tendency to excite" and is often likened to a spark burning along a fuse.

The action of ions is variable. Thus it has been found experimentally that solutions of sodium and lithium salts leave the normal electrical potential of the external surface of the muscle unchanged, while salts such as those of potassium, rubidium, and ammonia (which give other evidence of penetrating the muscle) produce an injury current or local negativity. Absence of penetration is indicated by failure to change the electrical potential on the external surface of the muscle. The salts of the alkaline earths (calcium, magnesium, strontium, barium, and other metals of the same group) and in part those of caesium are thus indifferent, i.e., do not change the electrical potential on the external surface of the muscle. Nevertheless, they do alter the properties of the tissue; thus magnesium salts exert a strongly anti-stimulating or narcotic action, and calcium and strontium salts in pure isotonic solution similarly render muscle resistant to stimulation. All of these effects are reversible if the exposures are not too prolonged; in other words restitution to normal may occur.

It may be helpful to state the type of electrical charge carried by the respective ions‡. Negative charges are carried by the anions (Cl, SO_4, HCO_3, etc.); cations (Na, K, Ca, Mg) bear positive charges.

When ions, with their electrical charges, move at different rates Osterhout uses the term "diffusion potentials"†. He found the mobility of potassium ions to be far more rapid than are those of sodium. He considers the potassium ion to be particularly important because it produces large diffusion potentials and high conductance through its high mobility[8].

Elsewhere in his Harvey Lecture, Osterhout says: "Until these potassium ions leave the protoplasm (aided by the reversal of current which normally occurs after the negative variation) and any structural changes are repaired, the protoplasm is said to be in a refractory state: when this is over it returns to its resting stage. But if potassium ions remain in the protoplasm or just outside of it they may produce some of the phenomena commonly called 'fatigue' (e.g., the next negative variation will be less marked because there is less potential difference of the protoplasm so that its loss produces less change)." Lillie believes that the loss of irritability during the refractory period is consistent with the hypothesis that the surface layer breaks down or is otherwise altered during stimulation, for the reason that semi-permeability, by means of which electrical polarization is achieved, is apparently essential for stimulation. Any temporary decrease in the state of semi-permeability must, therefore, involve a lessening of irritability*.

It may be pointed out that the mobility of these ions is very rapid,—quite rapid enough to achieve the quick changes in electrical potential that take place in the heart.

The application of the foregoing biochemical and physiologic data to the heart appears to be reasonable. Osterhout and Hill[9] state that the successive variations observed in the experiments on the cells of the aquatic plant, Nitella, recall those observed in the heart and in certain cases in nerve. Clark[10] puts forward the hypothesis that "the activity of the heart is dependent upon the semi-permeability of the cell to electrolytes". Lillie writes: "In cases of automatism, like that of the heart, the rhythm may be regarded as determined by periodic stimuli furnished by processes within the cell. Since all these phenomena may occur, or undergo modification, in response to changes of environmental condition, all are to be considered under the general conception of stimulation†."

Osterhout predicted that whenever he could reduce the potential difference at any point to zero (or reverse it) and retain this condition for a time he should be able to make a Nitella cell "produce successive negative variations like a heart muscle". He was successful in achieving this, both by the application of 0.05 m. potassium chloride to the cell and by killing one end with chloroform instead of applying potassium chloride. His published illustrations look like electrocardiograms with a succession of ventricular complexes,—Q.R.S.'s and T waves‡.

The gist of the foregoing is that living protoplasm possesses an electrical charge of potential dependent on the ions associated with it and that when at a given point the electrical po-

*Ref. 3, pp. 261 and 271.
‡Ref. 3, pp. 161 and 168.
†Ref. 5, pp. 173 and 175.

*Ref. 3, p. 297.
†Ref. 3, p. 265.
‡Ref. 5, p. 176.

tential is reduced or made negative it may be said to become active or excited. It is then electrically negative to the adjacent protoplasm and an impulse, which is an electrical current, is propagated as a wave along the contiguous resting protoplasm.

THE HEART

The heart contains certain specialized tissue,—the sino-auricular node, the A-V node, the bundle of His with its two main divisions and the Purkinje network. Normally the heart beat is initiated at the sino-auricular node and spreads wavelike over the auricles, whence it is conducted to the specialized tissues in the ventricles and thence to the ventricular muscle. It has been established that this process is associated with a change of electrical potential which can be recorded by the electrocardiograph.

The last edition of Starling's *Physiology*[11] summarizes the evidence because of which the neurogenic theory of the heart beat has been abandoned. I will mention but part of this evidence, namely that (1) the heart in the developing chick begins to contract at a date prior to the growth into it of nerve cells; (2) isolated cardiac muscle cells, cultivated outside the body, show rhythmic contractions; and (3) the velocity of conduction of the impulse is much slower in the heart than in a nerve*.

The sino-auricular node contains thin elongated muscle cells, striated, interlacing with one another in a plexiform manner, and surrounded by connective tissue. The remainder of the specialized tissue of the heart likewise differs histologically from that of the remainder of the heart; it is termed "lowly differentiated" by Starling. The A-V bundle and its primary divisions are enclosed in a layer of connective tissue which apparently isolates them from the adjacent muscle fibers. The Purkinje fibers or cells are well-known in the special tissue of the ventricle, but it appears to be less generally known that they have been found all through the auricles also[12]. In general it may be stated that much of the specialized tissue of the heart is isolated from the surrounding musculature up to its termination, where histological connections with the muscle fibers can be seen. More detailed histological description of the specialized tissue is available in numerous textbooks.

It is evident, therefore, that all that is needed to initiate a cardiac impulse is that the cells of the sino-auricular node become electrically negative† to the cells with which they are in contact. When this occurs they may be said to become activated, and according to the electrical laws described in the earlier part of this article, an electrical current is conducted over the heart.

It is known that the speed of the impulse varies in different parts of the specialized tissue of the heart. If the phenomena observed in myocardium, are similar to those existing in the nerve tissue, it may be that the speed is greater, the greater the calibre of the conduction tissue*. This is a possible explanation for the slower rate of the impulse in the functional tissues as compared to that in the specialized tissue in the ventricles.

CAUSATION OF THE INITIAL NEGATIVE ELECTRICAL POTENTIAL

An important and very natural question arises concerning what it is that reduces the electrical potential of the sino-auricular node and thus causes it to initiate an impulse or "propagated tendency to excite". The observations of Osterhout, cited above[5], contain the data from which this question may be answered. The blood returned to the heart contains ions whose electrical charges can so alter that present in the resting cells of the sino-auricular node. It is probable that the potassium ions are very important in reducing the electrical potential of these cells, because of the rapid mobility characteristic of potassium ions.

Are there potassium ions in the blood entering the right auricle? The answer is, yes. Van Slyke[17] states that hemoglobin has acid properties and combines with a certain amount of alkali in the form of potassium hemoglobinate. This combination appears to liberate its potassium ion freely in its function of acting as an important carrier of CO_2. It may equally readily liberate the potassium ions when it is passing through the heart.

How can the rhythmic production of impulses be explained, or in other words, after the sino-auricular node has discharged one impulse, how can it be induced to send off a succession of impulses? I have quoted from Osterhout† the description of the return of the electrical potential of the activated and discharged cells, to the potential of the resting state. Then the process of reduction of electrical potential of these cells is ready for repetition. There is no lack of suitable ions in the blood, for any used up are constantly replaced by new ones as the stream of blood flows into the heart.

*P. 700.

†Craib, in a recent monograph, presents a somewhat different theory of the electrical activity in the heart. He gives evidence in support of the statement that the customary conception of electrical negativity and activity are not synonymous. In fact he finds that part of the area in the active state may even be electrically positive. It does not seem essential to this paper to present a complete summary of Craib's monograph; he himself states (on page 27) that, in respect to the electrical phenomena observed in uninjured strips of muscle suspended in air, "the two hypotheses are . . . in close accord." However, in view of the evidence presented by Craib it would obviate objection if one wrote of the activity of the tissue being associated with a sufficient "change in electrical potential" rather than a state of "lessened electrical potential". The validity of some of Craib's conclusions has been questioned by Gilson[14] and Bishop and Gilson[15].

*Ref. 7, p. 63.

†Ref. 5, pp. 173 and 175.

WHY DO NOT ALL CELLS CONTRACT RHYTHMICALLY?

The answer to this question is that presumably, to a certain extent, all muscle cells possess this power, but that it is possessed to a higher degree by the specialized tissue of the heart. That statement in the latter part of the foregoing sentence does not, of course, tell us why.

It is possible that there are differences in permeability of different cell walls. The following quotation is pertinent. "One of the most remarkable feats of protoplasm is the construction of thin, non-aqueous, surface layers in contact with aqueous solution on either side. They are found not only at the external surface of its cells but also at those of nuclei, plastids, vacuoles, and other inclusions. By determining what passes in and out they not only control metabolism but they localize its processes and make possible the differentiation which accompanies development"*. Parsons quotes Loeb as stating: "We owe it to the calcium and magnesium salts of our blood that our skeletal muscles do not contract rhythmically like our heart"†. There are those[3, 5] who believe that the outer layer of cells contains an increased amount of lipoids,—lecithin, cholesterol, and cephalin,—and Osterhout asserts that lipoids have a high electrical resistance and are less permeable to ions, etc.

It is apparent that other ions than potassium are significant in the matter of cellular activation and impulse formation, and there may be factors of importance in differences in the membrane of different muscle cells. It would be helpful, of course, to obtain dependable explanation of why muscle cells differ in their tendency to initiate rhythmic contractions, but it is not essential to the acceptability of the explanation of the causation of the heart beat.

It is well known, however, that other parts of the specialized tissue of the heart possess the power to initiate contractions; for instance, if the sino-auricular node is put out of commission,—by processes occurring physiologically (vagal stimulation) or in disease, or by various experimental procedures,—the A-V node, or parts of the specialized tissue on the ventricular side of this, initiates contractions. Normally the latter are prevented from initiating contractions because they receive the impulse transmitted from the sino-auricular node, and contract in response to it, before they can themselves initiate an impulse. They are then said to be discharged, or their electrical potential is restored to that of the resting state (after a short period of being refractory) and any tendency to become electrically negative to adjacent cells must start anew. Before this reaches a sufficient degree, a fresh impulse, or electrical current, is transmitted from the sino-auricular node, and the process is repeated *ad infinitum*.

*Ref. 5, pp. 175 and 180.
†Ref. 2, p. 267.

It is clearly of some importance to obtain an explanation of the pace-making function of the sino-auricular node. In other words, what is it that causes the sino-auricular node to initiate contractions at a faster rate than do the other parts of the specialized tissue of the heart? So far as I am aware, there are few factual data on which to base an explanation of this point; I will, nevertheless, offer certain suggestions in the following: The problem of the chemical control of the sino-auricular node warrants experimental study.

The ions with their electrical charges conveyed by the blood from the coronary arteries may be considered to affect all parts equally. Pratt[18] cites Wiggers[19] as stating that the thin-walled auricular tissue receives its nutrient supply largely from the cavity of the auricle. The former writes: "We may conceive of the right atrium, with its associated pace-maker, as chemically governed, in part at least, by the content of fresh venous blood."

The cells of the sino-auricular node are separated from the blood in the right auricle merely by a thin layer of cells of the endocardium through which there are interstices, whereas it is known that the specialized tissue lower down in the heart (the bundle of His and its branches) is separated from the muscle and endocardium by a bursa which may be demonstrated by injection with India ink. There is, therefore, some anatomical support for the belief that the sino-auricular node is more readily affected by the ions carried by the venous blood entering this cardiac chamber. One recalls also that the primitive cardiac tissue of the embryo beats at a stage before either nerves or arteries have grown into it. The sino-auricular node, it would seem, continues this closer relationship to the adjacent blood stream.

COMMENT

An explanation of the causation and propagation of the heart beat is presented. It is appreciated that the data obtained from the literature of bio-chemistry and physiology are given briefly. It is probable that many physicians, who have hitherto not had occasion to read freely the literature pertaining to the nature of proteins, colloids, ions, electrolytes, etc., will find much assistance by reading on these subjects. It will doubtless be realized that this article contains little that is new; the literature of biochemistry and physiology contains much that appears to concern the nature of the heart beat. No attempt has been made to review such literature; merely some of that which has come to the writer's attention and has contained pertinent data is cited.

It has been pointed out that apparently very

similar phenomena exist in nerve tissue and in muscular tissue*; the functions of both are achieved by the operation of almost identical laws, namely, by changes in electrical potential. In the opinion of the writer this seems easier to believe than that an entirely different group of phenomena are needed for the function of nerves and muscular tissue.

Many students of biochemistry and physiology express the conviction that changes in the electrical potential are fundamental to many, if not most, of the vital processes† — nerve impulses, muscular contractions, glandular secretion, stimulation of special sense receptor, growth, movement of plant, etc.,—in the body. The phenomena associated with colloids, and with ions and their electric charges should not be ignored; they appear to offer the key to progress in the knowledge of much that goes on in the body.

If a clear understanding be obtained of the mechanism of the origin and propagation of the normal heart beat, it should prepare the way for progress in knowledge of abnormal states of the heart beat.

SUMMARY

Data selected from the literature of biochemistry and physiology are briefly presented.

It is known that the impulse associated with the heart beat is accompanied by a change in electrical potential, which is recorded by the electrocardiograph.

It is believed that the process which initiates the impulse is, in brief, a lowering of the electrical potential at the point, usually the sino-auricular node, from which the impulse starts. This lowering of the electrical potential in one part makes this part electrically negative to the parts to which it is adjacent. An electrical current, or the impulse, then flows to the adjoining cells, whose electrical potential is thereby lowered and the current flows, for the same reason, on to the next cells with which they are in contact; this is repeated until the current is distributed to the muscle cells which are thereby stimulated to contract.

The lowering of the electrical potential of the cells is believed due mainly to the action of potassium ions brought by the blood to the heart.

*Refs. 3, 5, and 9.
†Ref. 3, p. 315.

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RETROPERITONEAL PERIRENAL LIPOMA

BY LAWRENCE WECHSLER, M.D.*

RETROPERITONEAL perirenal lipoma is the term used to denote the fatty tumor arising from the perirenal fat. These lipomas often grow to enormous size and constitute a class of the largest abdominal tumors. Morgagni is accredited with having reported the first case of retroperitoneal perirenal lipoma, in the latter part of the eighteenth century[1]. In 1837, a picture of a kidney surrounded by an immense amount of adipose tissue, apparently arising from the perirenal capsule, was published in Rayer's Atlas[2]. Isolated cases have been reported since that time, and tumors weighing as much as forty to seventy pounds have been mentioned[1]. James C. Masson and E. J. Horgan reported twelve cases of retroperitoneal lipoma varying in weight from sixty grams to 9.75 kilograms[3]. However, tumors weighing over twenty-five pounds are exceedingly rare and are usually invaded by malignant changes. The following case of perirenal lipoma, the tumor weighing 11.1 kilograms, is added to the list:

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CASE REPORT

HISTORY:—A. L., a male patient, 65 years old, came to the office in October, 1925, complaining of pyrosis, belching and heartburn. Occasional constipation and nocturnal frequency were also present. The rest of the history was negative. Examination showed the patient to be an elderly male in apparent good health. His weight was 137 pounds and there had been no appreciable loss. The head, neck and thorax were negative. Abdominal ex-

amination was likewise negative, there being no masses, tenderness, rigidity, or any palpable organs. Rectal examination revealed a slightly enlarged and tender prostate. Blood pressure 130/80. Urinalysis was negative.

In February, 1927, the patient returned with the complaint of general weakness. Examination of the abdomen revealed a mass in the right hypochondrium, tender on palpation. The urine was negative for albumin; there were a few pus cells and a few red blood cells in the sediment. The patient refused roentgenological examination.

In June, 1928, the patient again returned, complaining of dyspnea on exertion and after meals. No gastric disturbances were noted. Weight 139 pounds. Blood pressure 130/80. The heart sounds were good in quality and regular in rhythm. No enlargement of the heart was noted. The abdomen was distended, the umbilicus protruding. No masses could be palpated. The urine was negative. Roentgenological study was again suggested, but the patient did not consent.

In March, 1929, the patient returned a third time because of distention of the abdomen. There was no nausea, vomiting or hemorrhoids. His appetite was good. Weight 146 pounds. Blood pressure 130/80. Abdominal examination revealed marked distention, with a bulging umbilicus. A mass was palpable in the lower right half of the abdomen, extending to the hypogastric region. It was hard in consistency but not tender. The liver edge was palpated at the level of the umbilicus. ROENTGENOLOGICAL EXAMINATION showed the heart and aorta enlarged; the right diaphragm very high and dome-shaped; the esophagus normal. The stomach was pushed up very high into the left upper quadrant of the abdomen. In the anterior-posterior position the pylorus was not seen, but in the oblique position the entire stomach was visualized. The contour was regular and peristalsis fairly active. In six hours there was a small residue in the stomach. In twenty-four hours the entire colon was filled and pushed to the left. *Barium enema:*—The medium filled up the left-sided colon and the transverse colon as far as the hepatic flexure. It could not pass beyond this region.

On October 21, 1929, the patient entered the hospital for the removal of ascitic fluid. The mass filled the entire right side of the abdomen and extended toward the left lower quadrant. The abdomen was greatly rounded and uniformly enlarged. The heart was normal. Blood pressure 130/90. The lungs were physiological. A complete blood count and the blood chemistry were normal.

OPERATION FOR REMOVAL OF ASCITIC FLUID:—Under novocaine solution, one-quarter per cent infiltration, a small two-inch incision was made through the lower right rectus muscle. On opening the peritoneal cavity, about a pint of bloody fluid escaped, but the abdomen was filled with a large bluish semicystic mass. The extent of the incision precluded extensive exploration so that its relations or connections could not be decided. It seemed, however, possibly to be connected with the liver or perhaps to spring from the retroperitoneal region. Aspiration of the mass in several directions yielded dark blood. There were a number of vessels traversing the mass and the dark bluish appearance of the mass itself suggested the possibility of blood content so that it was deemed unsafe to incise the mass for fear of encountering a hemorrhage which might not be easily controlled through the small incision, permission for an exploratory operation not having been obtained. *Preoperative diagnosis:*—Abdominal carcinomatosis. *Postoperative diagnosis:*—Abdominal carcinomatosis[4].

Following this operation the patient showed no signs of improvement and developed an irregular temperature curve ranging from 97 degrees to 101.7 degrees. This continued for a period of six weeks, when he suddenly developed a chill, thready pulse, and died within twenty hours.

NECROPSY:—The body was that of a well-developed and fairly well-nourished white male about the age of seventy. Permission for autopsy was authorized to enlarge the previous surgical incision of the abdomen and make a local study. The abdomen was markedly distended and a hard mass could be felt at the right upper and lower quadrants of the abdomen. The lower extremities showed marked edema extending from the hip to the toes. The original incision in the right hypochondriac region was enlarged and the abdominal cavity entered. The peritoneal cavity was filled with fresh and old hemorrhagic fluid, approximately a quart. All the abdominal viscera were pushed to the left side by an enormous tumor mass, which was located retroperitoneally, apparently originating from the perinephritic area. The liver was pushed high up toward the thorax. It was very much discolored, rather pale and firm in consistency, and had a mottled appearance, resembling that of a nutmeg liver. The spleen was slightly hypertrophic, the capsule firm and adherent. The cut surface presented a granular appearance with prominent pulp. The tumor mass was intimately adherent to the colon, part of the small intestines and the stomach. It was extremely hard to separate the adhesions. After careful dissection, the whole tumor mass was removed. The mass weighed twenty-five pounds, was multilobular and rather firm in consistency, except one of the lobules, located most anteriorly, which had undergone cystic degeneration, about the size of a watermelon, with resultant rupture. One corner showed necrotic areas where the rupture had taken place. The cavity was filled with numerous forms of blood clots, such as chicken-fat clots, firm brown clots, et cetera. Most of the clots were organized and adherent to the wall of the cyst. The lacerated area had a ragged, discolored margin. There were several fairly good-sized vessels visible. Microscopical studies from various parts of the tumor mass showed varieties of fat cells, mostly embryonic in type. There were occasional areas where fibroblasts were numerous. Other places showed different forms of degeneration, such as myxomatous, xanthochromatic changes. No evidence of sarcomatous changes was present. Sections from the kidney showed arteriosclerotic changes with numerous retention cysts.

Anatomical Diagnoses:—(1) Intra-abdominal lipoma originating from perinephritic fat; (2) intra-abdominal hemorrhage due to rupture of the lipomatous cyst; (3) arteriosclerotic kidney; (4) passive congestion of liver.

COMMENT

In 1881, LeCrampe-Loustau concluded that perirenal lipomas were practically never diagnosed clinically since they do not produce symptoms, and therefore, even if a tumor is diagnosed, perirenal lipomas cannot be distinguished from other tumors occurring in the same location. This case also presented difficulty in diagnosis as the symptomatology was vague at first and later, when the mass had grown large enough to be noted on examination, pressure symptoms were the only ones complained of. In this case, the tumor mass was present for more than two years and weighed twenty-five pounds, yet produced few symptoms other than discomfort due to weight. The patient's general appearance being good, and his appetite constantly excellent, the possibility of a benign growth was suggested; but due to the hardness of the mass and the age of the patient, malignancy was favored. Perirenal lipoma may only be suspected, then, upon the finding of a large tumor in the renal area and the ruling out by

urologic methods of hypernephroma, polycystic kidney, hydronephrosis, and perinephric abscess. Diagnosis is usually made at operation or necropsy.

The treatment is surgical. Early removal of the growth before it becomes too large to permit removal with fair safety, and before malignant change has been allowed to occur, is of the utmost import.

The prognosis in any case is grave. Recurrences after operation are frequent and these recurrent tumors tend to undergo malignant change.

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FRACTURE OF THE CAPITELLUM

BY FREDERICK S. HOPKINS, M.D.*

Anteroposterior before reduction.

FIG. 1.

Lateral before reduction.

FIG. 2.

*Hopkins—Assistant Surgeon; Director of Cancer Clinic, and Fracture Service, Springfield Hospital. For record and address of author see "This Week's Issue," page 1274.

Anteroposterior in acute flexion.

FIG. 3.

Lateral in acute flexion.

FIG. 4.

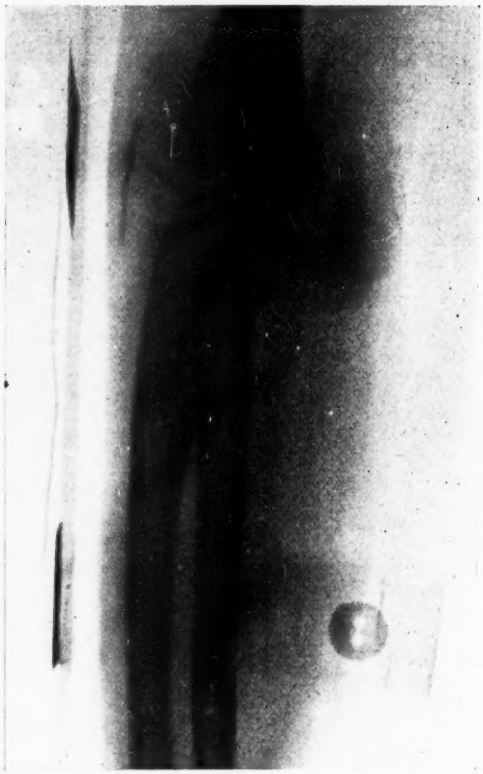
Anteroposterior after first attempt to reduce.
FIG. 5.

Anteroposterior immediately after reduction.
FIG. 6.

FRACTURES of the capitellum of the humerus are comparatively rare, a search of the literature revealing no duplicate of the following case.

R. S., a boy aged 5 years, was referred to me on August 10, 1931, by Dr. Daniel R. Wheeler with a history of falling off his velocipede and injuring his right elbow, probably by indirect violence through his extended hand and forearm. There was localized tenderness and swelling over the external condyle of the humerus and pain on motion at the elbow. No loose bone could be felt. X-ray examination by Dr. Howard L. Jackson showed a fragment about 1 x 1.5 cm. in size including the capitellum and tip of the external condyle displaced outside the joint with the fractured surface facing outward. The fragment could not be clearly visualized under the fluoroscope.

An open operation seemed indicated but an attempt was made to reduce the fracture immediately



Lateral immediately after reduction.
FIG. 7.

Anteroposterior eight weeks after reduction.
FIG. 8.

Lateral eight weeks after reduction.
FIG. 9.

under ethyl chloride general anesthesia. The maneuver was traction and adduction of the forearm and manipulation of the fragment. After two attempts the small fragment slipped into position. For fear of disturbing the position it was immobilized in extension.

After one week active motion was commenced and the elbow was held in gradually increasing flexion by moulded plaster splints. The splints were omitted at four weeks at which time the range of motion was from 45 to 140 degrees. Eight weeks after the injury his mother reported that he had been playing football. At this time the range of active motion was 55 to 170 degrees and of passive motion 45 to 175 degrees. X-ray examination showed callus formation with a small area of new bone formation on the anterior surface of the distal end of the humerus. On December 3, the range of active motion was from 50 to 175 degrees while that of his other arm was 45 to 180 degrees. Pronation and supination were normal. There was full return of strength and he did not notice the slight limitation of motion.

This case is a rare type of fracture probably caused by the impact of the head of the radius on the capitellum. It also illustrates the value of attempting a closed reduction even where open operation seems necessary.

REMOVAL OF SEPARATED UPPER EPIPHYSIS OF THE RADIUS*

BY FRANK H. BAEHR, M.D.,† AND LOUIS E. HATHAWAY, JR., M.D.†

THE center of ossification of the upper radial epiphysis appears at the fifth year, according to Gray's Anatomy[1]. Ossification is complete at the sixteenth year, and the consolidation of the epiphysis with the diaphysis occurs in the seventeenth year. This sequence is remarkable because the ossification center of the upper radial epiphysis is the last to appear among the long bones, while this epiphysis is the first of all to unite with the shaft of a long bone.

A study of the literature shows that injuries involving the elbow joint are very common, especially in children, but that separation of the upper radial epiphysis is comparatively rare. Cotton[2] states that "separation of the upper radial epiphysis is a rare accident, occurring only in small children; even in them it is less common than a fracture in this region. Possibly there may be a later interference with growth, probably very rarely".

No previous case reports have been found wherein the separated epiphysis has been removed in a child; and because of its effect on the future growth of the radius and the function of the elbow joint, the following case history is of interest.

*Read at the meeting of the Springfield Medical Association, November 30, 1931.

†Baehr—Member of the Surgical Out-Patient Department, Springfield Hospital. Hathaway—Member of the Fracture and Orthopedic Out-Patient Department, Springfield Hospital. For records and addresses of authors see "This Week's Issue," page 1274.

CASE REPORT

B. S., an eleven-year-old white girl, on July 28, 1926, while standing on a rock about a foot high, suddenly slipped and fell, striking the outer side of her elbow (with her arm in flexion). Pain and disability of the elbow joint immediately followed the accident. She was taken to Dr. Baehr's office the following day.

Physical examination was negative except for the left arm. The left elbow revealed right angle flexion in extreme pronation, with moderate swelling, especially marked over the region of the external condyle of the humerus and radial head, where it was exquisitely tender. An attempt at passive motion showed about three-fourths limitation of both flexion and extension of the elbow and supination was not only extremely painful but strongly resisted.

Roentgenograms (Figure 1) taken directly after the examination revealed a complete separation of the upper radial epiphysis, which was lying parallel to the shaft of the bone and at right angles to its normal position.

On July 31, 1926, at the Springfield Hospital under ether anesthesia, manipulation was attempted under the fluoroscope without noticeable improvement. The patient was moved to the operating room, where the arm was prepared as follows: clean shave, ten-minute scrub with tincture of green soap, alcohol, 3½ per cent iodine, alcohol and mercurochrome.

Operation: A longitudinal incision was made over the head of the radius, exposing the joint capsule.

through which the freely movable epiphysis could be palpated. A large needle was inserted through the capsule and into the loose epiphysis. Then a small opening was made in the capsule so that the epiphysis shelled out easily. (Figure 2.) All joint

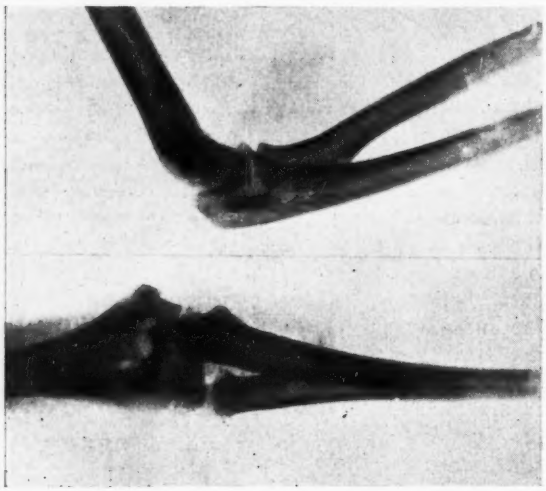


FIG. 1. In the above-mentioned case a separation of the epiphysis of the upper end of the radius is seen, with the epiphysis lying parallel with the shaft of the bone and extending upward from the outer surface.

motions were tried and found to approach normal. The joint capsule was sutured with continuous chromic catgut; the skin edges were approximated with interrupted silkworm sutures; and the arm placed in acute flexion with a Sayre dressing.

Postoperative History: The patient was dis-

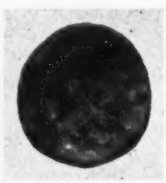


FIG. 2 (a). Antero-posterior view of the epiphysis after removal.

FIG. 2 (b). Lateral view of epiphysis after removal.

charged from the hospital on August 4, 1926. One week postoperatively, motion was begun and three days later the skin sutures were removed. Then hot soaks and massage were employed with continued passive motion. At the end of three and one-half weeks after operation, all motions of the elbow joint approached normal without pain in extreme supination. Complete restoration of function was brought about after two weeks of intensive physiotherapy treatment.

Clinical Course: The patient was seen on March 12, 1928, at which time all motions of the elbow joint approached normal without pain; the operative scar was non-tender—a good clinical result. Roentgenograms (Figure 3) were reported as follows: "Growth of the bone (radius) appears normal and there is a widening of the head of the left radius comparable to the head of a normal case. The character of the bone is good and the joint surfaces are smooth."

On March 26, 1930, the patient returned for a

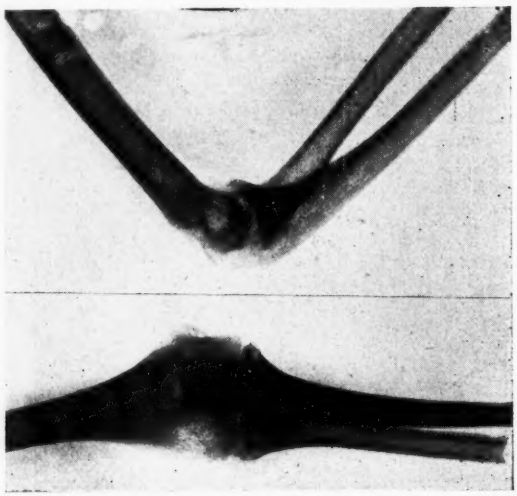


FIG. 3. In the above-mentioned case the original films taken at the time of the accident were reviewed, showing the epiphysis of the radius separated and lying at right angles to the shaft of the bone.

Plates taken today show the growth of the bone to be normal and there is a widening of the head of the radius comparable to the head in a normal case. The character of the bone is good and the joint surfaces seem smooth.

checkup, upon request. Comparison of both arms by measurements showed no appreciable difference in length.

On October 9, 1931, she was seen again and photographs and roentgenograms were taken. Measurements were made with the arms abducted and per-

FIG. 4.

pendicular to the body from the edge of acromium to the tip of the middle finger and were found to be 29 6/8 inches for the left arm and 29 7/8 inches for the right, showing a negligible difference in the length of both arms. The roentgenograms (Figure 4) show the left radius to be approximately 3/16

of an inch shorter than the right, which is about the thickness of the radial head of the unaffected arm. The photographs (Figure 5) illustrate the full excursion of motion of both elbow joints.

Discussing epiphyseal separations in general, Keen[3] states: "In the majority of injuries of this type, the epiphyseal cartilage drags with it more or less of the osseous tissue of the diaphysis." We have found this to be true in other cases, but in this specific case the roentgenograms and later examination of the shelled-out epiphysis showed clean, smooth edges.

FIG. 5 (a).

Textbooks give little or no information of upper radial epiphysis separation. Poland[4] published a list of seven cases in 1898, in four of which no details are given, and the diagnosis in the remaining three was made on physical examination alone. Included in this series was the case of Moullin[5], as follows:

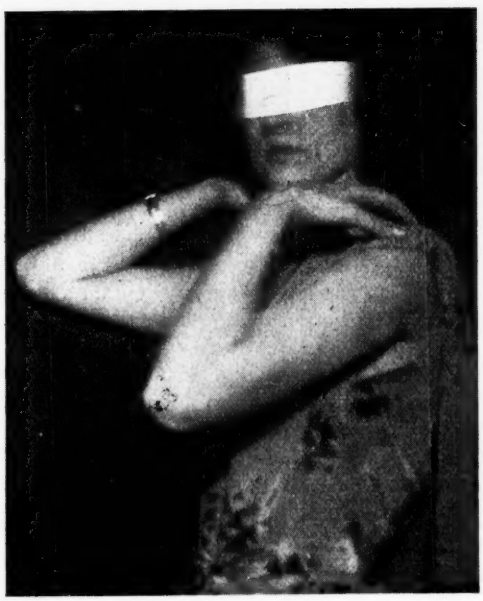


FIG. 5 (b).

A boy of thirteen years fell between a beam and a platform and sustained lacerations of the skin and muscles of his arm. A primary amputation was done above the elbow joint; the specimen at the London Hospital Museum demonstrates the upper radial epiphysis clearly detached at the epiphyseal line and partially in the grasp of the orbicular ligament. The diaphysis of the radius projecting behind was not otherwise injured, but the orbicular ligament was torn. The synovial membrane of the joint retained its connection with the radius.

Up to 1900 this case of Moullin's is the only reported proved diagnosis. Of a series of twenty-six cases of injuries to the head and neck of the radius Stoecklin[6] reported one case of upper radial epiphyseal separation, which occurred in a boy of seven years, who fell from a sled, striking his forearm against an iron rod and producing a compound fracture of the upper third of the ulna plus a separation of the upper epiphysis of the radius. Roentgenograms showed the epiphysis with a small attached fragment from the inner side of the diaphysis, lying free in the outer posterior part of the elbow joint. This patient was treated conservatively with heat, passive motion, and massage with resulting good motion except for a slight defect in supination.

Broca[7] mentions four cases he has seen at operation, but gives no details. In 1925 Montgomery[8] published the case of a patient of the same age as ours, in which he replaced the separated epiphysis by open operation in the orbicular ligament, where it seemed to fit snugly and firmly; the elbow joint could be moved freely while the epiphysis remained in position and rotated with the shaft of the radius. Three years later an examination revealed that there was no deformity or interference with the motion of the arm. Roentgenograms showed that the epiphysis had united with the shaft and a comparison of the measurements of the two radii showed the injured radius to be one cm. shorter than the radius of the other arm.

The mechanism involved in producing this epiphyseal separation is a force applied to the outer edge of the epiphysis with the elbow in flexion, causing it to flip out of the orbicular ligament. This type of injury could only occur with the elbow in flexion so that the epiphysis in moving could get by the capitellum of the humerus. Mechanically, Montgomery likens it to the act of raising a box cover by pressure downward and outward on the hinge edge. Beck[9] states that it may be produced by holding up or swinging a child by the arm, while Willard[10] mentions a violent jerk on the arm of a child as a possible cause.

DIAGNOSIS

A positive diagnosis of this type of injury cannot be made without roentgenogram examination and then only in children over six years

of age, when the center of ossification has appeared in the epiphysis. It seems to us that, with injuries about the elbow as common as they are in children, upper radial epiphyseal separation is probably more frequent than has been previously suspected, and that in children it should be looked for whenever tenderness and painful mobility of the radial head with or without deformity is found.

TREATMENT

There is no standardized treatment for this type of injury. Some men, like Stoecklin, have reported good clinical results with conservative treatment, as immobilization, heat, and passive motion; others, like Montgomery, recommend open operation with replacement of the separated epiphysis in the orbicular ligament; and of course, with the result obtained in this case, we feel that, if an open operation is to be done, removal of the separated epiphysis is a safe procedure.

The chief problem arising in the treatment of this type of injury, as with separation of any other epiphysis, has been the question of possible interference with the future growth of the shaft of the bone. We feel that our case substantiates the fact that the greater part, if not all, of the growth of the radius comes from the lower radial epiphysis, whose ossification center appears at the second to third year of life and does not unite with the shaft until about the twenty-fourth year.

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CANCER EDUCATION IN BOSTON

BULLETIN NO. 1, FROM THE EDUCATION COMMITTEE ON CANCER OF THE BOSTON HEALTH LEAGUE, JUNE, 1932

As a means of making the facilities for the early diagnosis and treatment of cancer better known in Boston, the Boston Health League organized an Educational Committee on Cancer in June 1931. This Committee has been meeting regularly throughout the year and functioning to arrange educational meetings and distribute literature about cancer.

Its major activity centered about the large Public Cancer Meeting which was held in Symphony Hall, April 6, with over 1200 persons attending—mostly lay people. Five hundred letters and over 3,000 programs were sent out in advance of the meeting.

During the month of May, evening health education meetings on "Cancer" were held in eight Health Centers throughout the City. The Committee furnished the speaker for each of these meetings. The audiences were composed of lay persons of various nationalities and the questions asked at the close of the meetings indicated great interest.

The Committee realizes that many persons are still reluctant to talk about cancer, and that until the public overcomes this reluctance, little progress will be made toward early diagnosis and cure. It believes that the best way to overcome this hesitancy is by spreading information about the subject, and will, therefore, furnish speakers without charge to groups wishing general information about cancer. At the office of the Boston Health League, 43 Tremont Street, there is a sample file of cancer literature, much of which is published in several foreign languages. This literature may be ordered for free distribution.

If you wish a speaker, pamphlets, or information regarding the City's resources for cancer treatment, please write or telephone the Education Committee on Cancer, Boston Health League, 43 Tremont Street, Lafayette 6564. Anna C. Palmer, M.D., Chairman; Mrs. Adolph Ehrlich, Mary R. Lakeman, M.D., Miss Eleanor E. Kelley, Miss Mildred E. Kennedy, Francis X. Mahoney, M.D., Miss Edith E. McCarthy, Rev. Thomas R. Reynolds, Harry A. Russell, Channing Simmons, M.D., Wilson G. Smillie, M.D., Shields Warren, M.D., Frank E. Wing, Margaret H. Tracy, Secretary.

DIABETES ANTITOXIN IN TORONTO

A group of Toronto medical research workers has been engaged recently in the study of the toxin or toxoid which may influence the behavior of diabetes. This research has been based on the assumption that a virulent type of the staphylococcus microörganism, commonly found in boils, carbuncles and abscesses, is responsible for a diseased condition of the pancreas that prevents its normal functioning. Production of the staphylococcus toxin and antitoxin at the laboratories was described at the meeting of the Canadian Public Health Association and the Ontario Health Officers' Association in Toronto during the last week of May.

The Toronto medical group includes Dr. S. Laird Alexander and Dr. H. McPhedran, throat specialists, and Dr. Joseph A. Gilchrist and Professor C. H. Best, associates of Dr. F. G. Banting in the discovery and clinical use of insulin. Besides studying the effect of the staphylococcus on the pancreas more than a year ago, some of the research workers began culturing the discharges of the nose sinuses with a view to discovery of the causative organism. The suggestion is made that certain facts are not yet worked out so that there may be no embarrassment to the group engaged in these studies which might result from attempts to apply the theories by individuals less well organized.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18241

IS ASTHMA A CAUSE OF DEATH?

MEDICAL DEPARTMENT

A German organist seventy-six years old entered complaining of severe productive cough of five days' duration. He was unable to answer questions for more than two minutes without becoming delirious. The history was obtained from a daughter.

He had never been strong and vigorous, and for fifty years had had a cough with more or less yellow sputum. In his youth he was suspected of having tuberculosis, but the diagnosis was never confirmed. He always had had about four "heavy colds" every year, with increased cough and sputum. He had had eczema on either his hands or his ears for sixty years. He had slight dyspnea and wheezing on exertion. Three years before admission he was ill in bed for four weeks with an acute respiratory infection,—fever, increased cough and much sputum. After a month at the seaside he felt as well as before the illness. Nine months before admission he had more dyspnea and wheezing on exertion, felt very tired and began to lose weight. Six days before admission the cough became violent and he began to raise large amounts of sputum. He felt very weak, and went to bed. Under medical treatment the cough did not improve. Violent coughing caused pain in his chest. He had no fever. The day before admission he became delirious and had difficulty in breathing. During the past year he had lost considerable weight in spite of a good appetite.

There was no known familial disease. One daughter had had rheumatic fever and chorea.

He had smallpox as a college student. For years he had had marked constipation. He had frequent gaseous eructations and slight discomfort after meals. For three years his appetite had been abnormally large. He had rare slight tingling of the fingers.

He entered the Emergency Ward with a good color, a strong pulse with frequent extrasystoles —every five beats—and a blood pressure of 150/70. The pupils were highly myopic, otherwise normal. His breathing was rather labored, with prolonged expiration and wheezing. He was somewhat irrational and talkative, and wanted to go home. The knee jerks were equal and somewhat sluggish. The chest showed marked hyperresonance. The lungs were filled with sonorous and sibilant râles. He was raising small amounts of purulent sputum at frequent intervals.

Examination in the medical ward showed the following additional findings. He was thin and coughing frequently. The eyes were prominent, the eyeballs hard. The fundi showed vitreous opacities and large crescents, all thought to be due to myopia. There was a defect of the left nostril with slight obstruction. There was slight tenderness over the left antrum and frontal sinus. The apex impulse of the heart and the percussion measurements could not be made out because of hyperresonance. There was a scratchy systolic murmur at the third left interspace transmitted to the apex. There were numerous extrasystoles. The artery walls were palpable and tortuous. The lungs showed, besides râles and hyperresonance, prolonged expiration. The tactile fremitus and voice sounds were normal. The liver edge was just palpable. The anus was red and raw externally. Examination showed very tender hemorrhoids. The prostate was somewhat enlarged. The pupils reacted sluggishly to light and accommodation. The reflexes were normal.

The amount of urine is not recorded. The specific gravity was 1.026. There was a slight trace of albumin at the single examination, and a slight trace of sugar. The sediment showed one to three white cells and one or two red cells per field. The blood was not remarkable except for 11,000 white cells the day of death. A Hinton test was negative. A blood culture showed no growth. The non-protein nitrogen was 25 to 34.

The temperature was 100.4° to 99.4°, the pulse 91 to 119. The respirations were 29 to 13 (morphia).

X-ray examination on the way from the Emergency Ward to the medical ward showed no definite evidence of disease of the sinuses. There was slight thickening of the mucous membranes in the region of the ethmoids and frontals. Examination of the lungs showed both hilum shadows abnormal, especially the right, along the right descending bronchus. There were also heavy streamers extending up towards both apices. The apices were not well visualized. The remainder of the lung fields showed no abnormality. The trachea was deviated slightly to the right at the level of the clavicle. The right border of the heart was not visualized. There was no definite evidence of active disease in the lungs.

The patient was disoriented and noisy the night of admission, with frequent cough. He was given one sixth grain of morphia subcutaneously the morning of admission and three

more doses that night, also syrup of hydriodic acid. The following morning he was comatose, with greatly slowed respirations. The blood pressure was 150/90 and the pulse good half an hour before death. That morning he died.

Clinical Discussion

BY MAURICE FREMONT-SMITH, M.D., AND AUBREY O. HAMPTON, M.D.

Dr. Fremont-Smith: What are the diseases of the chest that are confused with tuberculosis in youth? One is sinus disease with secondary chronic bronchitis or pneumonitis that may be secondary to that. A great many soldiers in the army were thought to have tuberculosis but were found to have sinus infection with a very chronic secondary bronchitis. Bronchiectasis is sometimes extremely difficult to tell from tuberculosis in young people. I think we are dealing in this case with bronchiectasis. I know of no other condition in which a man could remain well for so long and have a large amount of sputum and cough. In youth it is very hard to tell, especially if the bronchiectasis is in itself rather mild and there occasionally are patches of bronchopneumonia that extend from the bronchiectasis, often unilateral in type. Of course bronchiectasis itself is usually bilateral. I think a very safe rule is that if there are large amounts of sputum and you do not find tubercle bacilli after frequent examinations, the patient does not have tuberculosis. Of course there are plenty of cases where we get small amounts of sputum in which we do not find tubercle bacilli although the patient does have tuberculosis. Could he have had lung abscess? Not for this long period of time. He either would have died of sepsis or it might have disappeared, as abscesses of short duration are known to do. The story so far suggests that he had bronchiectasis and that he had bronchopneumonia as an extension or as a metastasis, one might say, from the bronchiectasis.

"For three years his appetite had been abnormally large." We know then that his gastrointestinal tract was normal. That is, if he had the thing we think of at seventy-six, cancer, his appetite would have failed, not increased. There are two diseases in which the appetite does increase. One is hyperthyroidism, the other diabetes. There is no evidence for either of those.

The sonorous and sibilant râles make of course a careful examination of the lungs very difficult. It is possible occasionally to pick up dullness. It may be possible to find characteristic râles in localized areas which would suggest pneumonia; but it is discouraging from the point of view of finer diagnosis to find a chest full of râles.

We know that pneumonia, bronchial or lobar, can cause marked delirium. Could there be here any other cause for this mental symptom, assuming for a moment that he did not have pneumonia? We get metastases from lung abscess to the brain, giving brain abscess. I do not know whether we do get them from bronchiectatic cavities or not. That is something to bear in mind as a poor possibility.

I did not know vitreous opacities could be due to myopia. Hard eyeballs would suggest glaucoma. That would be possible in a man of seventy-six. I should like to know if the anterior chambers were shallow.

The heart examination would go with fever and the fact that he was seventy-six years of age.

I do not see anything in the physical examination to help us very much. We know he has something more than asthma, more than emphysema, more than bronchiectasis. If he has had bronchiectasis or any serious lung infection for fifty years he should have clubbing of the fingers. He is not described as being cyanotic.

Miss Painter: It is definitely recorded that he did not have clubbing of the fingers.

Dr. Fremont-Smith: The albumin and sugar in the urine are probably not of importance.

We have to fall back, as we often do, on the x-ray examination to make a diagnosis of the intrathoracic condition. My guess is that there was more than was found by physical examination, that he had some areas of consolidation, and I should expect to find bronchiectasis at least.

Dr. Hampton: This is not the usual seven foot chest film. The diaphragm is usually high when the film is taken at a short film-target distance. Here the diaphragm is at the level of the twelfth rib. The report describes heavy streamers going down to the right base and increase of the lung roots, most marked on the right. Part of this dullness at each side of the heart is due to calcification of the cartilages. It looks much worse than it really is. The trachea deviates to the right. There is dullness at the apex and a ring area there that looks a little like a cavity. He has similar dullness on the left. The right apex is definitely smaller than the left and the trachea is displaced to the right side, so that you can see that there is chronic infection at the right apex which has left scarring and retraction of the lung, and also a similar process on the left side.

Dr. Cabot: Might that be tuberculous?

Dr. Hampton: I think it is evidence of old tuberculosis. I do not know whether that is a cavity or not. I think we doubt it usually.

Dr. Cabot: What would you say as to the evidence of bronchiectasis?

Dr. Hampton: I should not like to make a diagnosis on that film. In the presence of emphysema such as that apparently is, I think

we are not justified in laying too much stress on the lung markings, because we can nearly always say they are prominent throughout the chest. If we had a better examination it might be possible to differentiate dilated bronchi and blood vessels.

DR. FREMONT-SMITH: Can this all be emphysema?

DR. CABOT: He ought not to have sputum.

DR. FREMONT-SMITH: Exactly.

I do not know what he died of. He did not die of heart disease. He could not have died of anything we see in that chest. He might have had, as any man of seventy might, a cerebral hemorrhage, which would account for his mental symptoms and the very slight rise in temperature that we get as a result of internal bleeding anywhere. I am going to stick to my original diagnosis of bronchiectasis, and that is all I can say. Can you help me, Dr. Cabot?

DR. CABOT: If we did not have the x-ray I should say bronchopneumonia. I do not think we ought to rule it out simply because we do not see it.

DR. HAMPTON: Patients at postmortem usually have bronchopneumonia anyway.

A STUDENT: Do you think he had a brain abscess?

DR. FREMONT-SMITH: I do not think we have any reason for saying he had. We certainly have no physical signs.

A STUDENT: In a man dying in coma and with slow respirations isn't increased intracranial pressure likely?

DR. FREMONT-SMITH: The respirations of course were down anyway. He had a great deal of morphine that night, one sixth grain three or four times. That would bring the respirations down. I do not see that we have any evidence.

A STUDENT: What is the evidence for pneumonia?

DR. FREMONT-SMITH: Death in a man of seventy-six with prolonged chest symptoms. I have not made that diagnosis, but I think it is probably right.

DR. CABOT: I think that is the commonest cause of death in bronchiectasis.

A STUDENT: What is the rationale in giving so much morphia to a man with so much sputum?

DR. FREMONT-SMITH: To rest him. In a patient coming in with marked heart failure the important thing is to give that man a rest. I think that is also true of pneumonia. What that patient needs is rest rather than to get rid of a little more sputum.

DR. HAMPTON: Can a man die of asthma like that?

DR. TRACY B. MALLORY: Yes, but I never heard of a man with asthma going fifty years without a single paroxysm of dyspnea.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Bronchial asthma.

DR. MAURICE FREMONT-SMITH'S DIAGNOSES

Bronchiectasis.
Pneumonia?

ANATOMIC DIAGNOSES

1. *Primary diseases.*

(Bronchial asthma.)
Chronic bronchitis.

2. *Secondary or terminal lesions.*

Arteriosclerosis.
Emphysema.

3. *Historical landmarks.*

Acute and chronic rheumatic endocarditis of the mitral valve without deformity.
Healed chronic pulmonary tuberculosis.
Chronic fibrinous pleurisy.
Diverticula of the sigmoid.
Adenomatous polyps of the colon.
Melanosis coli.
Hyperplasia of the prostate.
Chronic prostatitis, slight.
Cholelithiasis.
Submucous lipomata of the cecum.

PATHOLOGIC DISCUSSION

DR. MALLORY: We found at autopsy a very marked degree of chronic bronchitis and nothing else of any great significance. There was considerable emphysema, not at all an extreme degree, not the amount of emphysema that would be apt clinically to cause death. There was very slight dilatation of the bronchi, but one has almost to stretch a point to call it bronchiectasis. The dilatation is not very marked. He did have a healed pulmonary tuberculosis at the apex, but there was no evidence of activity.

He had a definite though unimportant rheumatic heart disease involving the mitral valve.

He had a variety of other things which cannot have had anything to do with the symptoms, such as multiple diverticula of the sigmoid, adenomatous polyps of the large intestine, hyperplasia of the prostate, a few gall stones and several lipomata.

I think essentially he died of chronic bronchial infection, which is rare without an extension of the infection into the alveoli with a fairly obvious pneumonia at the time of autopsy. There was none here. I have seen two other cases quite similar, with acute and chronic bronchitis, no pneumonia and death. So I feel that a person can die simply of acute bronchitis on top of years of chronic bronchitis such as this

man had. Usually those cases have fairly well marked emphysema, as again he did. One case with almost exactly the same symptoms and the same anatomic findings was a woman under fifty, which is rather unusual. Emphysema of course is a disease of old people, like chronic bronchitis, but occasionally it can occur in fairly young people.

DR. CABOT: Was the tuberculosis at both apices?

DR. MALLORY: It was at the right apex only.

DR. FREMONT-SMITH: Do you get a brain abscess as a complication of bronchiectasis as you do with lung abscess?

DR. MALLORY: I do not remember having seen it. I do not know what the textbooks have to say about it.

DR. FREMONT-SMITH: What would be the difference in anatomy—that is, how do you distinguish between abscess and bronchiectasis?

DR. MALLORY: Sometimes it is very difficult to distinguish between abscess and bronchiectasis. Typical bronchiectasis is a multiple lesion which may be limited to one lobe or may be present in several. Perhaps the commonest distribution is in both lower lobes; the second commonest is restriction to one lobe. In this instance there is often definite bronchial obstruction above the point of dilatation, thus accounting for the localization. Lung abscess is more apt to be single, though of course it may be multiple too. The presence or absence of epithelial lining in the cavity does not absolutely settle the issue. It is always present in bronchiectasis; it may or may not be present in an abscess cavity. Most commonly an abscess is lined only by granulation tissue, but occasionally the bronchial epithelium does grow down into the cavity, particularly in the healing stages. It is not uncommon to find the two lesions together. If there is a single abscess near the apex of the lung it is very common to find bronchiectasis in the base of the lung. The pus from the abscess presumably drains down into the dependent bronchi, and these may eventually dilate into quite sizable cavities. If we were given merely a single cavity, however, we might have great difficulty in saying whether it were abscess or bronchiectasis.

CASE 18242

MULTIPLE LAPAROTOMIES FOR ECHINOCOCCUS CYSTS

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. JOHN L. NEWELL*: This is the case of a forty-nine year old Italian who first came into the hospital fifteen years before his last entry. At his first admission he had a laparotomy and removal of an echinococcus cyst. He had been in the hospital eight times in all before his final entry, and had four other laparotomies with removal of various cysts.

This time he came in complaining of pain in the epigastrium. The liver was palpable and irregular. He was operated upon again with a diagnosis of echinococcus cyst. An abscess was drained in the abdominal wall and the cyst removed.

Course in the hospital. He began by having fever immediately after the operation. This ran for three or four days. The wound became septic and was opened under gas. Soon after that he became more septic and ran a typical picket fence temperature. He finally became jaundiced and very anemic.

This is simply the story of a man who had multiple operations for echinococcus cysts. He finally died of liver insufficiency and sepsis.

CLINICAL DISCUSSION

DR. LINCOLN DAVIS: I have not much to say about this case. I did most of the operations. My recollection is that the cysts were widely disseminated from the start. They were scattered throughout the peritoneal cavity. Under those conditions it is almost impossible to get the last. If one small cyst is left others will form. The original cyst was probably in the liver, and ruptured with the escape of many daughter cysts which became implanted throughout the peritoneal cavity.

Dr. Means asked if x-ray affects them. Dr. Van Gorder has had a good deal of experience in China. He says he tried x-ray treatment out and is convinced that it has no effect. Dr. Holmes did give this man thorough x-ray treatment and we found active daughter cysts afterwards. The effective treatment is to remove all the lining wall of the cysts without contamination of the peritoneal cavity. If you can remove all the lining without contamination you can cure the disease. If the lining membrane cannot be removed, the treatment is to swab the cavity with strong solution of formalin, which is very efficacious.

DR. GEORGE W. HOLMES: These x-ray films are largely negative. On this film the diaphragm is high on both sides. There is no evidence of echinococcus disease in the chest. This film was taken after operation. There are changes in the lungs, but they are not characteristic of echinococcus. The three distinct pictures we get with echinococcus cyst depend on the stage the cyst is in. If it is an unruptured cyst it is a perfectly round shadow like a tennis ball. If it has ruptured into the lung, the appearance resembles lung abscess.

I am not sure whether that is the last film of the chest we have or whether this condition

*Senior interne on the East Surgical service.

is of echinococcus origin or not; I am more inclined to think it is pneumonia.

DR. DAVIS: Was it taken after operation?

DR. HOLMES: Yes.

The films taken of the urinary tract show nothing remarkable at all except an area which might represent pressure, but even then I do not think one would be justified in saying it is due to pressure from a tumor in the kidneys. In some cases the outline of an abnormally shaped kidney can be made out. I think most of these cases do show an abnormally shaped kidney, which is possibly of more diagnostic value than change in the shape of the pelvis of the kidney. The masses in the abdomen are not visible. These cysts rarely show calcification except when pneumoperitoneum is used. We did have one case years ago where we were able by pneumoperitoneum to show the cysts very well.

As to the question of treatment, what we might be able to do would be to destroy the egg. The egg should be quite sensitive to radiation and should be easily destroyed. To make the treatment effective we ought to treat the patient before the first operation. That we have never done. The idea of treatment would be not of course to destroy the cyst, but to sterilize it. We have not of course a great deal of proof that we can sterilize them, but theoretically it should happen to all the eggs of an animal of that type. In one case I sent an irradiated cyst to the pathologist, who fed it to dogs. None of the dogs developed the disease.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Echinococcus cyst of the abdomen.
Sepsis of the operative wound.

ANATOMIC DIAGNOSES

Multiple echinococcus cysts of the liver, spleen and small intestine with perforation and infection of one of them.
Purulent abscesses of the liver,—pylephlebitis or possibly infected cysts.
Hyperplasia of the spleen (Mediterranean spleen).

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: I think probably the most interesting part of this case to most of us is the gross specimen, which as you can see from the size of the basin required to contain it is far too large to pass about. The liver is simply filled with cysts and weighs over 5 kilos. There is a very large cyst in the splenic pedicle. The spleen itself is much enlarged, but not from echinococcus disease. Microscopically it shows only diffuse fibrosis, and I fear we shall have to be content with classifying it as a "Mediterranean spleen," very well known in Italians. Perhaps it went back to the malaria. There were a few small scattered cysts along the small intestine. We found none in the lungs. In the liver, in addition to the cysts there was a very definite infectious process which I will show you on the slides.

The first slide is from the unruptured uninfected cyst, showing here the typical laminated membrane, completely acellular. Here are numerous daughter cysts with scolices within them. One of the larger cysts was connected with a perforation in the stomach and had become infected, so that all evidence of its origin has disappeared as far as microscopic examination is concerned. There is no laminated membrane left. It is lined only with granulation tissue. There is little doubt however that this is an echinococcus cyst which became secondarily infected. Scattered through the liver are very numerous small purulent abscesses. It would be difficult to say with certainty that those might not be infected small cysts. I think it is much more probable however that he had a terminal pylephlebitis with multiple abscesses throughout the liver tissue, and that in all probability that was the immediate cause of death.

A PHYSICIAN: I should like to ask whether this man had any eosinophilia; also whether he had an echinococcus fixation test.

DR. NEWELL: At his second and third entries he had 12 per cent and 8 per cent eosinophiles. No increase was found at any of the later entries. An echinococcus fixation test at the second entry was negative. That is the only one recorded.

DR. DAVIS: It seems to me that a very interesting feature of this case was what Dr. Mallory just spoke of as the apparent perforation of one of these cysts into the stomach. I think that is very unusual. Was that a cyst of the liver that had perforated into the stomach?

DR. MALLORY: It is difficult to tell whether the cyst perforated into the stomach or an ulcer of the stomach perforated into the cyst.

ERRATUM

IN Case 18232 of June 9, two lines at the beginning of the Pathologic Discussion on page 1219 were omitted. The lines are as follows:

DR. TRACY B. MALLORY: This case seems to have about all the complications that one well — — — —.

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POSTGRADUATE INSTRUCTION IN FRACTURES

DURING the Great War the treatment of fractures of the long bones was tremendously improved, a natural result of a wealth of material under perfect supervision. Although it took many months, or even more than half the duration of the war, to standardize the treatment of fractures on the field and in the various hospital units, it became obvious, particularly to the American surgeons, that by this method only could proper results be obtained. The very simple order of "splint them where they lie" saved countless lives and innumerable limbs.

The medical profession has been slow to standardize the treatment of injuries in civil life, comparable to those in war. Of course, there are many reasons for this, the lack of Government or State control probably being foremost among them. Individual war-trained doctors splint their fractures and send them to the hospitals, from force of habit in many instances, but even this influence has not reached more than one-tenth of such injuries that occur today. With the innumerable and increasing automobile accidents, with fractures of the long bones resulting therefrom, our problem today is really not unlike that of the war. What can be done to improve this situation? Shall we appeal for Governmental standardization and have the medical profession feel that they are being dictated to? Can we, through educational methods, reach the doctors of the state carrying to them instruction in the emergency handling of fractures? To be sure, certain progress has been made along this line, as now in New York City and Philadelphia where, due to the efforts of a few individuals interested in such a problem, every ambulance has been equipped with Thomas leg and arm splints so that now these patients are splinted at the place of their injury and transported to the hospital without increasing the deformity and overriding and without compounding their fractures. This is so rigidly enforced that ambulance men would lose their jobs if they should bring an unsplinted fracture into the hospital. The improvement in results in cases thus transported is striking when one visits the hospitals treating such injuries in these cities today.

Certainly we should carry this work further than the Municipal Governments have seen fit to do in these two cities. It does not seem reasonable to expect the State to take on such a problem. The various State Societies should be the leading spirit in directing this type of education. Ways and means must be found to carry to the local societies in New England the simple methods of emergency splinting and the simple methods of treating fractures of the long bones by modern, up-to-date, adequate methods. It is easy to speculate what economic saving would result from such propaganda. The doctors who are called to treat such injuries are serious in their intentions to do the best for the patient that their present knowledge permits. We have evidence that they wish to improve their methods of treatment. We are sure they would be receptive to time-saving devices which would give their patients better results.

THE ANNUAL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY

THE organized functions of the Massachusetts Medical Society for the 1931-1932 year were finished with the one hundred and fifty-first session of June eight, nine and ten.

The interest in the affairs of this Society was never exceeded if one can judge from the spirit

shown in all of the sessions of this anniversary. Beginning with the elaborate and well-conducted clinics at the Boston City Hospital on June 8, with an exceptionally large attendance, and ending with the annual oration by Dr. S. B. Woodward on June 10, the interest throughout was sustained and the attendance most gratifying. The registration amounted to nearly twenty-five per cent of the membership.

The weather was favorable and economic conditions apparently did not operate to any large extent in keeping the Fellows from attending the exercises. Perhaps the comparatively better health conditions throughout the State may have given freedom to some who otherwise might have been detained at home with serious cases.

The attendance at the Council Meeting was large and there was a feeling of satisfaction that the problems, which had called for consideration at previous meetings, had been met in a judicial spirit and progress made toward unanimity of purpose in adjusting conditions which might have led to serious differences of opinion if dealt with in any narrow or prejudiced fashion.

We believe that we represent the sentiment of the Society in stating that the creation and work of the Committee on Public Relations will stand as one of the constructive achievements in the history of the Society.

The annual banquet which is the especial social function was well attended and enjoyed. The cordial greetings of our English confreres were extended by Mr. G. R. Girdlestone of the Royal College of Surgeons and head of the Winfield Hospital at Oxford, England. Two public health officials, Dr. J. M. Baker, Health Commissioner of Alabama, and Dr. Felix J. Underwood, Health Commissioner of Mississippi, discussed the problems of public health in its relation to medicine and paid enthusiastic tribute to the work of the Massachusetts health administration and especially to the Commissioner for his devoted service to the people of our Commonwealth.

Dr. Charles F. Wilinsky, Deputy Commissioner of Health of Boston, explained the local problems of the capital city of the State and emphasized the necessity for coöperation and support by the medical profession.

Mr. Joseph A. Parks, Chairman of the Massachusetts Industrial Accident Board, explained the workings of the law relating to compensation for accidents and gave assurance of fair dealing with the profession.

Mr. Robert G. Dodge, Counsel of the Society, presented figures showing that alleged malpractice suits against physicians are less frequent today than formerly and referred to the greater number of cases requiring disciplinary action for irregularities of behavior on the part of lawyers as compared with objectionable and unethical conduct of physicians. He explained the procedures of the grievance committee of the bar association and the recourse to the courts in appropriate cases; the belief was expressed that a similar method would be an improvement over that provided in the statutes for dealing with erring physicians.

The Society was especially honored in having as its guest, Dr. Dean Lewis, President-Elect of the American Medical Association, and Professor of Surgery at Johns Hopkins University. Dr. Lewis, in referring to his service in the World War, spoke of the decline of the morale which was evident in the war period and the apparent purpose of many now to secure compensation for conditions which have no relation to military service. He felt disturbed because of the unsound proposals for building and equipping veterans hospitals which will not be required later especially since there are available a sufficient number of beds in civil hospitals, supplementing government institutions, to meet the needs of veterans. He discussed the costs of medical care briefly, explaining that when people are sick they demand the most expensive accommodations and other luxuries which, added to the increasing expense of scientific medicine, account for the costs of illness in many cases.

The Rev. Arthur Pratt of Greenfield was the last speaker. He made an elaborate plea for the united support of the medical and clerical professions in promoting and maintaining clean living to the end that progress in morals, health and civilization will be secured thereby.

The last session of this annual meeting was given over to Dr. Samuel Bayard Woodward of Worcester, war-time President of the Society for the years 1916-1919, who delivered the annual address under the title of "The History of Smallpox in Massachusetts."

This oration is the consummation of the work of Dr. Woodward during and subsequent to his occupancy of the presidential chair, for then and in the following years he has labored consistently and persistently to induce the people of Massachusetts to adopt adequate measures for the prevention of smallpox. His address is a complete compilation of the important facts relating to smallpox in Massachusetts.

He was honored by a large audience which spontaneously rose at the conclusion of the address in appreciation of its quality and the work of the author.

Taken all in all this meeting will stand as a demonstration of coöperation of the officers of the Society and the Sections together with the stimulation of large and appreciative audiences.

The official report of the Secretary will set forth the details of the State Society and Council meetings in a subsequent issue.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MALLORY, FRANK BURR. A.B., M.D. Harvard University Medical School 1890. Professor of Pathology, Harvard Medical School and Pathologist, Boston City Hospital. Editor, American Journal of Pathology. His subject is "Cirrhosis of the Liver." Page 1231. Address: Boston City Hospital, Boston.

CLUTE, HOWARD M. B.S., M.D. Dartmouth Medical School 1914. F.A.C.S. Surgeon, Lahey Clinic. Assistant Surgeon, New England Baptist Hospital and New England Deaconess Hospital. His subject is "The Operative Mortality in Goitre." Page 1240. Address: Lahey Clinic, 605 Commonwealth Avenue, Boston.

PEIRSON, EDWARD L. JR. M.D. Harvard University Medical School 1925. Assistant in Urology, Massachusetts General Hospital. Assistant Urologist, Salem Hospital. Urologist, Lynn Hospital and North Shore Babies Hospital. His subject is "The Reasons for Unsatisfactory Results in the Treatment of Calculi of the Upper Urinary Tract." Page 1243. Address: 33 Summer Street, Salem, Mass.

HODGKINS, EDWARD M. M.D. Tufts College Medical School 1915. F.A.C.S. Assistant Professor of Surgery, Tufts Medical School. Assistant Visiting Surgeon, East Surgical Service, St. Elizabeth's Hospital. His subject is "A New Method of Treating the Indirect Sac in Inguinal Herniorrhaphy." Page 1249. Address: 12 Bay State Road, Boston.

ARCHIBALD, ROBERT E. M.D. Dalhousie University. State District Health Officer, Massachusetts Department of Public Health. Address: 18 Bassett Street, Lynn. Associated with him is

FEEMSTER, ROY F. A.B., M.D. Johns Hopkins University School of Medicine 1924. Dr. P.H. 1925. Assistant Director, Division of Communicable Diseases, Massachusetts Department of Public Health. Address: 546 State House, Boston. Their subject is "A Small Milk-Borne Outbreak of Septic Sore Throat." Page 1252.

REID, WILLIAM D. A.B., M.D. Harvard University Medical School 1909. F.A.C.P. Assistant Professor of Cardiology, Boston University School of Medicine. Physician, Massachusetts Memorial Hospitals. Member, Evans Memorial. Cardiac Consultant to Newton Hospital and Waltham Hospital. His subject is "The Causation and Propagation of the Heart Beat." Page 1254. Address: 510 Commonwealth Ave., Boston.

WECHSLER, LAWRENCE. M.D. University of Pittsburgh 1914. Chief in Gastro-Enterology to the Montefiore Hospital, Pittsburgh, Pa. His subject is "Retroperitoneal Perirenal Lipoma." Page 1259. Address: 3401 5th Ave., Pittsburgh, Pa.

HOPKINS, FREDERICK S. A.B., M.D. Harvard University Medical School 1918. F.A.C.S. Assistant Surgeon; Director of Cancer Clinic, and Fracture Service, Springfield Hospital. Consulting Surgeon, Shriners' Hospital for Crippled Children. His subject is "Fracture of the Capitellum." Page 1261. Address: 146 Chestnut St., Springfield, Mass.

BAEHR, FRANK H. Ph.B., M.D. University of Vermont Medical College 1922. Member of the Surgical Out-Patient Department, Springfield Hospital. Address: 20 Maple St., Springfield, Mass. Associated with him is

HATHAWAY, LOUIS E. JR. A.B., M.D. Harvard University Medical School 1928. Member of the Fracture and Orthopedic Out-Patient Department, Springfield Hospital. Address: 20 Maple St., Springfield, Mass. Their subject is "Removal of Separated Upper Epiphysis of the Radius." Page 1263.

MISCELLANY

AN HONOR TO DR. HARVEY CUSHING

An honorary degree of Doctor of Science was awarded to Dr. Harvey Cushing by Northwestern University at the commencement exercises June 13, 1932.

THE PRESIDENT OF THE CONNECTICUT STATE MEDICAL SOCIETY

At the Annual Meeting of the Connecticut State Medical Society, held May 25-26, Dr. William A. La Field was elected President. He resides in Bridgeport and is a graduate of the New York Homeopathic Medical College and Flower Hospital.

Dr. Thomas P. Murdock, the Secretary of the State Medical Examining Board, was elected a delegate to the New England Medical Council to fill the position made vacant by the resignation of Dr. Robert L. Rowley. Dr. George Blumer of New Haven is the president of this Council.

DR. HENRY SIEGERIST SUCCEEDS DR. WILLIAM H. WELCH, PROFESSOR OF THE HISTORY OF MEDICINE

Dr. Henry Siegerist has been appointed to and accepted the chair of Professor of the History of Medicine at Johns Hopkins University to succeed Dr. William H. Welch, who has resigned.

Dr. Siegerist has been the Director of the Institute of Medical History at the University of Leipzig. He is the author of "Man and Medicine: An Introduction to Medical Knowledge" and has recently visited the United States and delivered lectures at several universities.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1931 AND SEVEN YEAR AVERAGE

MONTH ENDING MAY 28, 1932

Diseases	1932					1931			
	Week ending May 7	Week ending May 14	Week ending May 21	Week ending May 28	Average cases reported for week corresponding to May 28 for past seven years	Week ending May 9	Week ending May 16	Week ending May 23	Week ending May 30
Cerebrospinal Men.	—	1	1	—	1	1	1	2	—
Chicken Pox	91	101	113	120	67	49	101	103	107
Conjunctivitis Inf.	2	—	16	11	2	6	—	4	—
Diphtheria	3	3	3	6	19	9	17	6	3
Encephalitis Epid.	—	—	—	—	1	—	2	—	—
German Measles	4	2	8	5	55	10	11	9	10
Influenza	20	7	3	3	5	7	2	2	2
Malaria	—	—	—	—	—	—	—	1	—
Measles	231	296	299	273	270	582	699	634	435
Mumps	51	73	84	71	50	58	67	63	80
Paratyphoid Fever	1	—	1	1	—	—	—	—	—
Pneumonia (Broncho)	34	40	23	24	23	18	24	22	24
Pneumonia (Lobar)	46	34	35	47	28	41	46	17	21
Poliomyelitis	—	—	—	—	—	—	—	—	1
Scarlet Fever	106	97	106	127	56	47	42	54	35
Septic Sore Throat	2	5	1	4	—	2	3	1	1
Tetanus	—	—	1	—	—	—	—	1	—
Trachoma	—	—	1	—	—	—	—	—	—
Trichinosis	—	—	—	—	—	1	4	3	1
Tuberculosis (Pul.)	40	32	21	36	41	23	20	28	19
Tuberculosis (O. F.)	4	3	4	2	3	3	—	4	2
Typhoid Fever	—	2	—	2	2	—	5	2	1
Undulant Fever*	—	—	1	—	—	—	4	1	—
Whooping Cough	74	129	96	96	49	52	31	47	47
Gonorrhea	23	21	37	27	27	14	14	38	31
Syphilis	55	45	47	28	26	49	23	46	25

*Made reportable August 14, 1928.

Remarks: No cases of Asiatic cholera, glanders, plague, or yellow fever during the past seven years.

ECONOMIC CONDITIONS AS RELATED TO HEALTH

It is interesting to note, as set forth by the New York City Department of Health, that there has been a continued decrease in the general death rate in that city even though unfavorable economic conditions have prevailed for the two past years. Tuberculosis, for example, has continued to be a decreasing factor in mortality and morbidity rates, for the number of reported cases in 1930 and 1931 is less than the records of 1929. This condition is especially surprising because the examination of school children during the past six years has shown an increase in malnutrition in this group from 13.5 per cent to 20.5 per cent. The fear of Commissioner Wynne is expressed in the inference that this apparent increase in malnutrition will be followed by an increase in morbidity unless economic conditions improve.

On the other hand the increase in cancer deaths among the population groups of forty years or more is marked.

A THEORY RESPECTING THE GENESIS OF CANCER

Mr. Sampson Handley, in his book on "The Genesis of Cancer", has presented the theory that lymph stasis may be the cause of cancer and he claims to have found that this condition is at least frequently present in carcinoma and that it has not been proved to be absent in any case. Although there may be three alternatives: lymph stasis may be the essential cause; it may be merely a contributory cause or a constantly associated phenomenon unre-

lated to the causation of the disease, he is committed to the belief that time will show that lymph stasis is the essential cause of cancer.

These statements are found in the *British Medical Journal* of January 20, 1932, and quoted in the *Bulletin of the American Society for the Control of Cancer.*

AN HONOR TO DR. FRANCIS G. BENEDICT

Dr. Francis G. Benedict, Director of the Nutrition Laboratory of the Carnegie Institution of Washington, has been given the honorary doctorate in medicine on the occasion of the 350th anniversary of the University of Wuerzburg. The caption on the diploma, which states very specifically the basis of the award and is phrased most happily is as follows:

Die Medizinische Fakultät der Universitat Würzburg
ernennt durch einstimmigen Beschluss
Herrn
Professor Dr. phil. Francis G. Benedict
der, abwahl Chemiker, Seine ganze Arbeit in den Dienst der Medizin stellte und diese besonders auf dem Gebiete des Stoffwechsels durch glänzende Methoden und durch zahlreiche Untersuchungen von bleibendem Wert bereicherte, zum
Doctor medicinae honoris causa
Würzburg, 12, Mai 1932.

RECENT DEATHS

KEEN—Dr. William Williams Keen of Philadelphia died in that city June 7, 1932. He was born in Philadelphia January 19, 1837. He graduated from Brown University in 1859 and the Jefferson Medical College in 1862, afterward lecturing on pathologic anatomy and being advanced to the Professorship of Surgery in his Medical Alma Mater. After a service of eighteen years in this last-named position he was made Professor Emeritus. He was elected an Honorary Fellow of the Massachusetts Medical Society in 1924 and had served as President of the American Surgical Association, the American Medical Association, the College of Physicians of Philadelphia, the Congress of American Physicians and Surgeons and several non-medical organizations.

In 1920 Dr. Keen presided at the Sessions of the International Congress of Physicians and Surgeons in Paris. His honorary degrees were received from Harvard, Yale, Pennsylvania, Brown, Toronto, Edinburgh and other universities. He was awarded the Bigelow Medal by the Boston Surgical Society in 1922.

He has occupied a position in the medical and scientific world exceeded by no one in this country and has been ready to exert his great influence in professional and civic affairs.

He married Miss Emma C. Borden of Fall River, Mass., in 1867. She died in 1886.

Four daughters survive him, Mrs. Walter J. Turner of Philadelphia, who has seven children; Miss Florence Keen, who made her home with her father; Mrs. George W. Handy of West Hartford, Vermont, and Mrs. Howard Butcher of Ardmore, Pennsylvania, who has six children.

Two descendants are interested in medicine. Dr. Norman Freeman is associated with Dr. Walter Cannon in the department of physiology of the Harvard Medical School and Dr. Walter Freeman is practicing medicine in Washington, D. C.

BOWMAN—Dr. Alfred Winthrop Bowman died June 7, 1932, at his home, 108 Pleasant Street, Arlington. He was born in Augusta, Maine, the son of Dr. Alfred W. Bowman. He graduated from the Harvard University Medical School in 1901 and had an office at 263 Newbury Street, Boston. Dr. Bowman is survived by a widow, Mary J., two sons, Alfred and Paul, and three daughters, Sister Ethel of the Jeanne d'Arc Convent, Milton, Annie and Miriam.

HOLMES—Dr. Harry Clinton Holmes died at his home, 47 Rutland Square, Boston, June 6, 1932, after a long illness. He was a member of the Massachusetts Medical Society from 1896 to 1911. Dr. Holmes was a graduate of Tufts College Medical School and Harvard Medical School. He was born in Charlestown August 10, 1858. There are no immediate survivors.

CROCKETT—Dr. Eugene Anthony Crockett died at his summer home in Ipswich, Massachusetts, June 13, 1932. He was a Fellow of the Massachusetts Medical Society, having joined in 1891.

He graduated from the Harvard University Medical School in 1891 and practiced Otology in Boston, occupying the position of Assistant Professor in that specialty at the Harvard Medical School. He was a major in the World War, serving with the Red Cross in Italy, Serbia, and elsewhere. He is survived by his widow, three sons, and a daughter.

DEATH OF TWO WORKERS IN THE TUBERCULOSIS FIELD

In the death of Dr. Elmer H. Funk of Philadelphia, the tuberculosis movement has lost a staunch friend and supporter. Dr. Funk died suddenly from angina pectoris on May 14. He was 47 years old. For several years he served as clinical professor of medicine at Jefferson Medical College, but last September he was appointed professor of therapeutics, succeeding the late Dr. Hare. In association with Dr. Thomas McCrae, dean of Jefferson Medical College, Dr. Funk was assistant editor of the Osler System of Medicine and was also the author of a textbook on diseases of the respiratory tract.

He was a former director of the National Tuberculosis Association.

Dr. Charles Hampson Jones died in Baltimore on April 11 in his seventy-fourth year. He was a charter member of the National Tuberculosis Association, and a member of the Board of Directors. He was a graduate of Johns Hopkins University and spent four years at the University of Edinburgh, Scotland. After his return to Baltimore in 1883 he was prominent as a teacher, practitioner, and for 14 years as Commissioner of Health of that city. Through his association with the Baltimore Dairy Council he was instrumental in bringing about many milk and food regulations. In a tribute to him Dr. William H. Welch said, "It is no exaggeration to say that he did more for the protection and promotion of the health of the people of his city than anyone throughout the entire history of the city."

NOTICES

CORRECTION

In the book review on "Normal Young and Its Everyday Problems" by Douglas A. Thom it was incorrectly stated that the book was the winner of the medal awarded by the *Parents' Magazine* to the best parents' book of the year. This is an error as the award was given to the book "Everyday Problems of the Everyday Child."

REMOVAL

Frederick H. Verhoeff, M.D., announces the removal of his office to the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

REMOVAL

Samuel Sandberg, M.D. announces the removal of his office from 300 Seaver Street, Roxbury, to 144 Columbia Road, Dorchester.

UNITED STATES CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces the following open competitive examination:

Medical Technician
(Bacteriology and Roentgenology)

Applications for the position of Medical Technician (Bacteriology and Roentgenology) must be on file with the U. S. Civil Service Commission at Washington, D. C., not later than July 19, 1932.

RADIO HEALTH MESSAGES

JUNE, 1932

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:20 P.M.

June

21 Community Public Health
28 Communicable Diseases

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:30 P.M.

Health matters of public interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

REPORTS AND NOTICE OF MEETINGS

THE SOUTH END MEDICAL CLUB

The regular monthly meeting of the South End Medical Club was held on Tuesday, May 17, at 12 o'clock noon.

The speaker was Dr. N. A. Nelson, Assistant Director, Division of Communicable Diseases, Massachusetts Department of Public Health.

GONORRHEA AND SYPHILIS

N. A. NELSON, M.D.*

On the basis of reports to the United States Public Health Service and on what is known as to the incompleteness of reporting, it is evident that gonorrhea is many times more prevalent than any of the common reportable communicable diseases and that syphilis is second only to gonorrhea. At least 1% of the population of this country is constantly under medical attention either for gonorrhea or for syphilis.

The control of these two diseases must be added to the responsibilities of the public health official and of the medical profession, for to be infected with either of these diseases has long since ceased to be the "special privilege" of the morally delinquent and has become altogether too frequently the undeserved burden of the innocent. So long as more than 10% of reported gonorrhea in the female is in girls from a few weeks to fourteen years of age, so long as at least half the women who have gonorrhea or syphilis are married women who have acquired their infections through marriage, so long as nearly 10% of syphilis is congenital, and so long as both diseases are predominantly diseases of youth, it will be our duty to bend every effort toward the control of their spread.

Since neither gonorrhea nor syphilis can be reported, as other communicable diseases, with the name and address of the patient, it becomes impossible for the health officer to proceed to their control through the use of ordinary administrative procedures. Mass quarantine and isolation are as impossible administratively and economically as they should be unnecessary if the patient is properly instructed as to his conduct. Unfortunately since public opinion makes the identification of the case

*From the Massachusetts Department of Public Health.

to the health officer impossible, it also makes impossible the usual search for contacts and the source of infection, and the physician has become not only the ultimate but the only health officer in contact with the case. Upon him alone lies the responsibility not only for keeping the patient under control but for discovering and bringing to medical attention those infections which may be related to the case in hand. If the physician will not accept this responsibility, he is in effect serving notice that so far as gonorrhea and syphilis are concerned he has little regard for the protection of the public health.

In the control of gonorrhea and syphilis more than in any other field of public health activity, there is need that the physician take active part. Health departments are often criticized for their actual practice of preventive medicine, in spite of the fact that through its various medical groups and societies the medical profession could have established itself (as well as the health department has done so) in the minds of the public as interested in the progress of preventive as well as curative medicine.

The health department provides every possible service for making diagnosis easier and a therapeutic armamentarium more readily available. It has even attempted the postgraduate information of physicians in up-to-date methods of procedure. It strives to teach the patient the importance of remaining under regular treatment. In short, it does everything that it can think of to encourage the practice of preventive medicine by the physician and to keep the practice of curative medicine where it belongs. In the control of gonorrhea and syphilis, except for the support of clinics for the treatment of indigents, there is little that the department of health can do except to assist the physician. It remains, therefore, to be seen whether or not the medical profession will seriously accept the responsibility for the control of these two diseases as it insists it should be permitted to do to the exclusion of the department of health.

We are convinced that compulsion and regulation have no place in the control of gonorrhea or syphilis and serve rather to drive both diseases still further under cover. The only control that is to be had over the patient, therefore, lies in the extent to which the patient is properly informed and adequately treated and in the extent to which new infections may early be brought to medical attention. To whom other than to the physician who sees the case and who alone has any contact with the case can we look, then, for the control of these diseases?

THE MASSACHUSETTS GENERAL HOSPITAL

A clinical meeting of the staff of the Massachusetts General Hospital was held in the Moseley Memorial Building on Thursday evening, May 19, 1932. Dr. George W. Holmes presided.

Dr. Hugh Montgomery presented a medical case. A forty-five year old Italian housewife was sent to the hospital by her family physician who had made the diagnosis of myelogenous leukemia. The only subjective symptom was dyspnea which had been moderate for two years and severe enough to confine her to bed for the previous two months. On physical examination the lungs were clear and the heart was not enlarged. There was a large firm mass in the left flank which extended down to the brim of the pelvis. The liver edge was felt about four centimeters below the costal margin. The white blood count was 190,000 with 87 per cent of the cells belonging in the myelocytic series. The majority were young cells, myelocytes varying between eight and seventeen per cent.

Treatment was instituted with Fowler's solution, four minims three times a day. There has been no evidence of gastro-intestinal irritation and the patient has shown marked clinical improvement. However the liver and spleen have not decreased in size and there has been no change in the blood picture. It will be necessary either to increase the dose of Fowler's solution or resort to radiation therapy in order to reduce the number of circulating leucocytes.

Dr. Claude E. Forkner spoke on "The Use of Arsenic in the Treatment of Diseases of the Blood." It is probable that no other drug has been more widely used for a longer period of time than has arsenic. More than two thousand years ago it was recommended in the treatment of asthma and at the time of Galen it was a very popular drug. It fell into disrepute largely because of its toxic effects and for many generations its use was prohibited by law. Fowler, in 1786, noticed that quacks were obtaining some remarkable cures with a secret preparation and had it analyzed. It proved to be an alkaline solution of potassium arsenite and tincture of lavender. Since then the preparation has been known as Fowler's solution.

There are reports of about twenty-five isolated cases of myelogenous leukemia treated with Fowler's solution in the literature between 1880 and 1904. At the latter date, radiation therapy was instituted and the use of arsenic fell into disrepute. During the past two years Dr. Forkner and his associates at the Boston City Hospital have studied the effects of Fowler's solution in twenty-two cases of chronic myelogenous leukemia. Administration of the drug, in adequate doses, is followed by marked symptomatic improvement, decrease in the size of the liver and spleen, lowering of the basal metabolic rate, lessening of the anemia, lowering of the white blood cell count, and partial or even complete disappearance of the abnormal cells from the circulation.

A typical case is that of a sixty year old farmer who had had the disease for two and a half years and had received ten x-ray treatments over the spleen during that time. The last treatment reduced the white blood cell count from 150,000 to 25,000 cells per cu. mm. but there was little clini-

cal improvement and the remission was short. He was then given Fowler's solution and in ten days the white blood cell count began to fall and soon reached 2500 cells per cu. mm. He was clinically well for four months but the leucocyte count began to rise after three months and reached 350,000. A second remission lasting three months was caused by the administration of a solution of potassium arsenite without tincture of lavender. This proved that the arsenic in Fowler's solution and not any constituent of tincture of lavender is responsible for the therapeutic result.

Of the twenty-two cases, nineteen have responded to arsenic therapy. Two were unable to take the drug because of gastro-intestinal disturbances and the third, a man in the fourth year of the disease, failed to respond to this or any other form of therapy. Remissions produced by arsenic usually last between three and four months and are associated with marked clinical improvement. There is no evidence, however, that the treatment is curative but it is very effective in the palliation of the disease.

The results in chronic lymphatic leukemia are not nearly so striking. Dr. Forkner has used it in three cases and in none has the count returned to normal although there has been a significant drop in each case. Arsenic is totally ineffective in the treatment of acute leukemia and, if anything, hastens the course of the disease.

Billroth in 1871 presented a case of lymphoblastoma benefited by Fowler's solution. A single case has been treated with the drug at the Boston City Hospital with marked symptomatic improvement and decrease in the size of the tumor masses.

Türk in 1904 reported a single case of polycythemia vera in which the red blood cell count fell from 9.2 to 7.8 million after the administration of Fowler's solution. No other cases have been found in the literature. At first thought it would seem illogical to use arsenic in treating this condition as the drug has for many years been recommended for various types of anemia. However, polycythemia vera and chronic myelogenous leukemia are similar in many respects and Dr. Forkner has used the drug in a number of cases. In each case it has caused a marked reduction in the red count, usually to normal, and there has been marked clinical improvement.

Before Minot and Murphy published the liver therapy for pernicious anemia Fowler's solution had also been used extensively for this condition. Two patients with pernicious anemia have been so treated at the Boston City Hospital. In each there was a definite reticulocyte rise, to seventeen per cent in one case and to forty-four per cent in the other. The drug is not recommended for the treatment of this disease, but was used simply to complete the study.

In using the drug it is important to give it regularly and to increase and decrease the dose slowly. There is considerable variation in the amount of the drug required. Treatment should be pushed until the therapeutic effect is obtained or signs of toxicity develop; these last are anorexia, restlessness, sleeplessness, and diarrhea. Dr. Forkner has not seen either hyperkeratosis or peripheral neuritis developing in any patient although some have taken the drug for nearly two years. Five of his thirty-five patients have developed herpes zoster. This complication is more likely to occur if the dose is increased too rapidly or the drug is given irregularly.

The apparent paradoxical action of arsenic of either increasing or decreasing the red blood cell count and hemoglobin concentration according to whether the patient has anemia or polycythemia and its effect in reducing the leukemic aspects in chronic leukemia and in lymphoblastoma were explained on the basis that arsenic in the form of potassium arsenite exerts two effects, dissimilatory and assimilatory. These effects are dependent largely on the state of the cells in the blood-forming organs. Arsenic exerts a dissimilatory or destructive action much more readily on young abnormal cells than on normal cells. On this basis its effect in the various diseases discussed is explainable.

The paper was discussed by Drs. George Minot, Francis Hunter, William Dameshek, and G. W. Holmes.

LAWRENCE CANCER CLINIC

Lawrence, Massachusetts, June 6, 1932.

To the Physicians of the North Half of Essex County:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, One Garden Street, Lawrence, on Tuesday, June 21, at 10 A.M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, and Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*
Committee.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

June 21—Lawrence Cancer Clinic. See notice above.

June 21-24—Catholic Hospital Association. See page 369, issue of February 18.

July 10 - August 12—The Second Intensive European Summer Course in Surgery, Gynecology and Obstetrics. See page 714, issue of March 31.

Autumn—International Postgraduate Courses in Berlin. See page 1169, issue of December 10.

September 19-22 — International Congress of Biliary Lithiasis. See notice on page 427, issue of February 25.

October 24-27 — American Public Health Association. See notice on page 1321, issue of December 31.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

BOOK REVIEWS

RECENT PUBLICATIONS BY THE NATIONAL TUBERCULOSIS ASSOCIATION

The National Tuberculosis Association has issued several new publications that will be of interest. They were shown on the book table at the annual meeting in Colorado Springs and the majority of them are for distribution through state tuberculosis and health associations.

What You Should Know About Tuberculosis (Spanish Edition). At the request of several affiliated associations a Spanish edition of this 32-page illustrated pamphlet has been printed.

Tuberculosis Nursing for Public Health Nurses, by Violet H. Hodgson, R.N., Assistant Director, National Organization for Public Health Nursing has been completely revised and is in a new size, 48 pages and cover. This booklet, which has proved its value to all nurses who require a sound tuberculosis nursing program is a textbook for every public health nurse and may be easily carried for ready reference in her bag. It may be purchased through your state tuberculosis or health association.

Books on Tuberculosis. The 1932 edition of this list of authoritative books compiled annually for physicians, nurses, patients, and executives has been published in a new and more convenient size with 24 pages. It has been brought up-to-date and contains an index of authors that will prove helpful in selecting books. Single copies will be sent upon request. If interested in quantities write to your state tuberculosis and health association.

The Journal of the Outdoor Life (Special Colorado Number). Extra copies for special distribution with "Summer 1932" instead of "June 1932" are ready for distribution. Single copies 25c each sold by the *Journal of the Outdoor Life*, 450 Seventh Avenue, New York.

Diversion for Cure Days. This is a new 4-page circular to advertise the *Journal of the Outdoor Life.* Attractively printed in green tint, this leaflet contains a general description of the magazine and also several comments from the readers, showing how they value the *Journal.* Write for free copies to the *Journal of the Outdoor Life.*

Advertising "*The Control of Tuberculosis in the United States*," by Philip P. Jacobs, Ph.D., a new 2-page descriptive leaflet has just been issued. A summary of the four parts is given and also a number of quotations taken from the many book reviews written by well-known health and tuberculosis workers. The book has been welcomed as a reference and textbook. Extra copies of the circular may be secured upon request.

Poster Service Supplement. This is issued but once this year and is for sale through the National Tuberculosis Association for $5.00. It contains 18 photographs of new posters and supplementary material for 18 agencies that issue posters. The Poster Service Book, up-to-date with the 1932 supplement, is $25.00.

The Health Talk. Its Place in Education, by Iago Galdston, M.D. supersedes the "Health Speaker's Handbook" and contains much valuable information on the psychology and mechanics needed for enlisting an audience's interest in health. For sale by the National Tuberculosis Association.

Robert Koch's announcement of his discovery of the tubercle bacillus, a translation of the original paper by Dr. and Mrs. Max Pinner.

Tuberculosis Mortality Among Young Women, the first of a series of social research studies. This study by Edna Nicholson of all deaths among young women in Detroit, case by case, was for the purpose of discovering what factors underlie the high death rate among this age group. It is for sale by the National Tuberculosis Association.

Health Conditions in North Harlem, the second of the social research series is a two-year study by a graduate student, a Negro, at New York University and covers not only the health conditions as shown by vital statistics, but gives also the social and environmental background of that group. It contains interesting and quotable health figures regarding the Negro. For sale by the National Tuberculosis Association.

The Cambridge Medical School: A Biographical History. By Sir Humphry Davy Rolleston. Cambridge University Press, 1932. ix + 235 pages. Price 15 shillings.

The author, Regius Professor of Physic in the University of Cambridge, has written a most interesting biographical history of the worthies who have preceded him in the teaching of medicine and surgery at this famous university. The life of each man is briefly considered and the general growth of the Medical School under the leadership of a series of brilliant physicians and scientists is depicted. The striking feature of the book is the excellent illustrations. Brief references are given to important biographical material, there is an excellent index and a complete list of the Linacre lectures from 1524 (?) to date. Such a volume is a first-class source book, as well as one of particular interest to the medical historian and to all who enjoy reading about the past masters of our profession.